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# Symposium

## Constitutional Causes of Orthodontic Failures*

### INTRODUCTORY REMARKS

J. CAMP DEAN, D.D.S., OAKLAND, CALIF.

THOSE of us who have practiced orthodontics, as our guest, Dr. Oppenheim, has, for thirty years or more, know what it means to encounter a case where nature does not respond to orthodontic stimuli readily. The case drags along for years, exhausting the patience of all concerned. Then finally, after a long term of treatment and retention, it relapses somewhat to the original condition.

In the beginning of orthodontics as a science, we were warned in the early textbooks about rickets and rachitic children, due to dietary deficiencies. Also models were shown of gross anomalies with undulated porous bone. The teeth would move readily but would not retain. This was due, it was stated, to constitutional disease, probably congenital syphilis.

Then came the extensive researches of Dr. Percy Howe with guinea pigs, rabbits, and monkeys, showing the collapse of dental arches when the animal was deprived of vitamin C. It was stated that the bone would return to normal when orange juice was given, but the terrible malocclusion would remain.

Still later came the well-known extensive research at the University of California Dental Department, under the supervision of Dr. John A. Marshall. A large number of monkeys were imported and treated and some of them were brought to normal conditions of health; then they were treated orthodontically, and results were observed under various conditions of diet. An effort was also made to show root resorption as a result of rapid tooth movement. This research was initiated by our Society and later jointly by the American Society of Orthodontists. The findings of those experiments were transmitted by Dr. Marshall to other Societies in the United States and throughout the world.

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Thus we have pointed out a few instances where the gross irregularity of the teeth is caused by constitutional disturbance. Many of us believe that these few instances do not tell the story; that there are many conditions which may not only cause the irregularity, but also make successful treatment impossible. How can the orthodontist recognize certain adverse constitutional conditions in children whom he is called upon to treat? Is he justified in proceeding with treatment when the evidence points to the fact that he is treating a symptom and that the cause of the anomaly lies in a disturbance of some organic function or malnutrition?

Dr. C. D'Alise of Italy writing in the October, 1940, number of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY states that the specialties of orthodontics and pediatrics should be combined as the solution. I can think of but one such specialist in the person of Dr. Harold J. Noyes of Chicago, head of the Orthodontic Department of Northwestern University. Few men find it practical to complete courses in both professions. Therefore, as a further warning before accepting orthodontic responsibility and treatment of subnormal cases we have brought you a conference of medical experts on various phases of the problem. They will volunteer such information as seems to them pertinent and discuss questions which may come before this conference.

# A SHORT REVIEW OF BONE AS WE UNDERSTAND IT BIOCHEMICALLY, HISTOLOGICALLY, AND PHYSIOLOGICALLY

JOHN A. MARSHALL, D.D.S., PH.D., SAN FRANCISCO, CALIF.

## CHEMISTRY OF BONE

BONE is an animal tissue, not vegetable, not mineral, which consists chemically of approximately 35 per cent by weight of organic matrix. This matrix is protein in nature for it is composed of finely interlaced fibers of one of the collagens designated as ossein. The remaining 65 per cent, which includes water, is inorganic, the solid portion of which consists of a crystalline structure very closely resembling two well-known minerals, dahllite and apatite. Neither the phosphate nor the carbonate exists in the free state, but always in crystalline combination with calcium together with very small amounts of magnesium and fluorine. In this connection it is interesting to note that the minutest variation in fluorine intake, whether of water or of food, produces extraordinary deficiencies in calcification in both bone and tooth formation.

## HISTOLOGY OF BONE

The normal histologic picture reveals a few scattered cells surrounded by hard intercellular substance and pierced at irregular intervals by Haversian canals, which contain blood vessels. Both inner and outer surfaces are covered by a specialized corrective tissue, a fibrous mat, the endosteum and periosteum.

No one can help but marvel at the structural design of bone. It is so adapted that it provides in health adequate support with the least amount of expenditure of weight and material. Under the stress of circumstances, this architecture can be changed very quickly; it is a matter of hours, rather than days or weeks. The ease with which these changes occur seems particularly remarkable when it is recalled that bone consists of only scattered cells; therefore, its metabolic rate under normal conditions is slow. In spite of this, there are constant changes going on. In fact, alterations in formation and reformation never cease, and are governed by causative factors which are both local and systemic in nature.

## PHYSIOLOGY OF BONE

There are three functions in which bone serves the body; first, to support it and to provide for its locomotion; second, to protect, the vital organs within the cranium and thorax; third to serve chemically as a storehouse for available calcium compounds, and by so doing to aid in the formation and coagulation of blood, in the maintenance of muscular tone and action, and in regulation of kidney function. All three of these are of equal importance, nor can they be considered separately but exist rather in an equal interdependent relationship to each other.

Professor of Biochemistry and Dental Pathology, University of California College of Dentistry, San Francisco, Calif. Now deceased.

Whenever the architecture of bone is studied, reference occurs to the Wolff Law which states in effect that every change in the form of bone, or its function, or both, is followed by corresponding change in the internal architecture and external conformation. This idea was cryptically expressed by John Murphy of Chicago when he said the amount of growth in a bone depends on the need for it. Thus the Wolff Law accounts in part for the pattern of work which may be performed by both osteoblasts and osteoclasts, for bone is constantly being built up and torn down in accordance with the muscular stress which it is called upon to withstand. But when disease develops, another property becomes manifest and stimulus of growth is withheld, or increased, or is even reversed as in the case of resorption, and again reversed as in apposition.

The exact mechanism by which calcium is deposited in the bone is still subject to great controversy. The early theory involving differences in tension of carbon dioxide with corresponding precipitation of calcium salts does not entirely explain the facts. During growth of bone there are continuous alterations taking place; tissue is built, torn down, and rebuilt again and again. To accomplish these purposes it is necessary that the tissue contain active cells, the osteoblasts, which in turn are governed both in rate of activity and in direction of function, by the enzymes of the phosphatase group. Furthermore it is known that building of bone requires a definite concentration of calcium salts precipitated in a suitable fibrous matrix. The second theory of bone growth has to do more particularly with the specific function of the osteoblast.

Agreement of the two theories is found in that both state that deposition can occur only in the presence of an adequate supply of bone minerals. The theories disagree as to whether the stimulus for building is chemical or cellular. It would appear that both theories may be sufficiently modified to form a new hypothesis in which the osteoblast carries the phosphatase to the osteogenic area. This is suggested in studies in rebuilding of new tissue in an area previously resorbed.

We, first as dentists, and specifically as orthodontists, are interested in studying bone changes and in assessing the value of those influences constantly at work, which may be favorable or inimical to its development. With these thoughts in mind your chairman has brought together this group to discuss the respective viewpoints of their own specialties in medical practice, those factors which may spell success or failure in the prognosis in some particularly difficult case.

# DENTAL STIGMAS OF CONGENITAL SYPHILIS

H. J. Templeton, M.D., Oakland, Calif.

WHEN Dr. Dean extended this invitation to me my reaction was twofold: First, appreciation of the honor but, second, apprehension in regard to the topic that he assigned me. If he had asked me to talk about actual syphilis of the maxilla it would have been fairly easy. We all know something about gumma of the palate, perforations of the palate, the caving in of the bridge of the nose following destruction by tertiary lesions. But, instead, he asked me to talk about the effect of congenital syphilis upon the development of the bones of the maxilla and middle third of the face and, therefore, its relation to the success or failure of orthodontic procedures.

I assure you that there is reason for apprehension when that subject is given to one, because there is practically nothing known about it. And I knew that very well. I wrote to Stokes, who has written the Bible in English as far as syphilis is concerned, to Parounagian of New York, who has been interested in this subject, to our medical libraries and to our dental libraries and secured very little information. Like Omar Khayyam, "Ever more came out wherein I went." However, for many years there has been a belief among dermo-syphilologists, especially those of the French school, that congenital syphilis does play some role in producing malformations of the maxilla. But it has simply been a belief with very little factual evidence behind it.

Stokes has said that about 5 to 6 per cent of all children born in the United States have congenital syphilis. That is too high a figure for this district, I am sure, because in arriving at it there was averaged in the high incidence of syphilis among the negroes of the South, where it may go up as high as 40 per cent. I am quite certain that in the general run of population here we will not have over 1 per cent who have congenital syphilis, and inasmuch as about half of those, according to figures, show dental stigmas, you may figure that you may possibly find dental stigmas of congenital syphilis in about one out of every two hundred of your patients.

There are many theories as to the pathogenesis of these stigmas. Some have felt that they occur because of the direct action of syphilis upon the endocrine glands and the indirect action of the perverted endocrine secretions upon the bone that is being formed. That is one theory. Another, according to Parounagian, is because syphilis has a well-known action on the calcium metabolism. But probably the best theory is that of a toxic action of syphilis upon bone centers at the time most important in the development of those centers.

There are two types of dental stigmas which are recognized as absolutely pathognomonic of syphilis: First, the true Hutchinsonian tooth, and second, the change that occurs in the first permanent molar known as the mulberry

Consultant, Dermatologist and Syphilologist, University of California, Cowell Memorial Hospital.

molar or the molar of Moon. In order to introduce my subject I am going to speak briefly about the theory of the formation of those teeth.

When a child is syphilitic in utero it is receiving during the months of its development its nutrition from its mother and is protected to a high extent by the mother's immune mechanism. However, when it is born it is immediately thrown upon its own defense mechanism, and at this time the spirochetes are particularly active in the liver and spleen, and the toxemia is at its height.

Now, the embryology of the Hutchinsonian tooth and the mulberry molar is such that they are in their most important period of development exactly when the toxemia of syphilis is at its height.

The central incisor teeth, the only ones that can show typical Hutchinsonian changes, develop from three lobes. These lobes are in the process of embryologic development in the first months of extrauterine life at the time when the spirochetic toxemia is at its worst. This is particularly true of the middle lobe. Since this middle lobe is mainly affected, the two lateral lobes grow downward more rapidly, leaving the middle lobe behind. This results in a single crescentic moon-shaped notching of the incisural edge of the tooth and also in the tooth being tapered from the broader gingival portion to the narrower incisural edge (so-called "pegging"). Likewise being hypoplastic the teeth are smaller than normal (microdontia) and, being smaller, are more widely separated (diastasia). These features of pegging, notching, microdontia, and diastasia are those of the Hutchinson tooth.

The one other typical syphilitic tooth is the "mulberry molar" seen only in the first permanent molar because this is the only tooth other than the central incisors which is developing in the first few months of extrauterine life when the syphilitic toxemia is severe. Let us consider the mechanism of the production of this mulberry molar.

Just prior to birth, while the child is being protected by its mother's immune mechanism, the enamel cusps of the first permanent molars are formed. They are laid down in a fairly normal manner because the syphilitic child is under its mother's protection. If the development of the tooth went on normally after birth the intercuspal substance would fill in between the enamel cusps and they would be forced outward to their normal position at the periphery of the tooth. But in the syphilitic child this intercuspal substance, embryologically due to be formed immediately after birth, runs into the intense syphilitic toxemia of this period and is stopped in its development. Later, as the child develops its own defense mechanism, the shank of the tooth develops more normally. So we have the picture of normal enamel cusps crowded together by a hypoplasia of the intercuspal substance and existing on a fairly normal base. These crowded together normal cusps give the tooth its "mulberry" appearance.

Other changes that occur in the congenital syphilitic are bossing of the frontal area, a widening of the space between the eyes, but a hypoplasia of the bones of the maxilla and the nose. Some of your authors, such as Nichalas, Massey, and De Pasquale, have said that the germinal anlage of the incisor bud and the other buds from which the middle third of the face develops are particularly susceptible to the action of the spirochetic toxemia. I checked with the Department of Anatomy at Stanford University and was told that that

particular area is also in process of ossification at the same time that the Hutchinsonian tooth and the mulberry molar are being formed. So it is a chronologic thing. The malformations of the Hutchinson tooth, of the mulberry molar, and the hypoplasia of the maxilla all can be explained by the coincidence of the intense syphilitic toxemia and the period of embryologic development of these structures.

There is another stigma that will be of some interest to you, and that is a caving in of the bridge of the nose. It occurs because of the same coincidence of period of development and syphilitic toxemia. I am not referring to the caving in of the nasal bones that occurs as a result of actual syphilitic destruction but rather to a parasyphilitic developmental dystrophy.

Parounagian of New York has for many years presented cases before the various New York medical societies claiming that a high-arched palate was quite indicative of congenital syphilis, the high arching and the crowding together of the teeth making a narrower maxilla. That is the original French idea. It would be explained on the same pathogenesis that I have just mentioned. Many of the New York men have disagreed with Parounagian very bitterly and have said that that is not correct, but at least in Parounagian's mind a high-arched palate and a narrow arch is at least quite suggestive of syphilis.

C. Morton Smith, in checking over 1,000 cases of congenital syphilis, came to the conclusion that an open-bite is suggestive of congenital syphilis. One author by the name of Iszard made the rather bold statement that open-bite occurs only in congenital syphilis. I doubt that very, very much, and I imagine you will all agree with me in doubting it. But the open-bite should cause us to search for syphilis.

If we admit that these parasyphilitic stigmas do occur because of syphilis, what can be done about them? What can be done to prevent them? Again, not a great deal is known. Stokes had one very interesting case. A diagnosis of congenital syphilis was made when a child was four months old. Stokes took x-rays and demonstrated most beautifully a typical unerupted Hutchinson tooth in that four-month-old child. Treatment was started immediately. The child was pronounced cured when he was five years old. Yet when the first permanent molars erupted, they were still Hutchinsonian.

Denny in his recent book on congenital syphilis made the statement that it is a fallacy to think that any amount of treatment will alter these dental stigmas. Cruckshank, writing in the proceedings of the Royal Society of Medicine, says this: "If treatment is begun shortly after birth, the dental stigmata do not occur." These are two diametrically opposed ideas. I imagine that the discrepancy there can be explained by considering the time the treatment begins. It is only my opinion (and I have not a thing with which to back it up) that if you took a congenital syphilitic child and began treating it the first week after birth before the syphilitic toxemia damaged it, you might prevent the development of Hutchinson teeth, mulberry molars, and hypoplasia of the maxilla. But I think that if you wait until the child is three or four months old, until ossification has set in, treatment would not prevent the dental stigmas.

What are we going to do about it if we take this pessimistic view? Several things. Permit me to wander away from dental fields and talk about syphilis

itself. The only way you can cause your orthodontic procedures to be more successful in the congenital syphilitic is to stop the syphilis before it develops. Syphilis can be stamped out in this country. The control of syphilis has been proved to be very practical in Sweden, and I am going to use that country as an example. Sweden has a population which is approximately equivalent to that of upstate New York, exclusive of New York City. In about 1915 Sweden had 5,000 cases of syphilis. Then they began a campaign for the control of syphilis, and in 1935 they had only 400 cases of syphilis. That represented a drop from 5,000 to 400 cases in twenty years. Now, at the same time (1935) upstate New York, with a population almost identical to that of Sweden, reported 40,000 cases of syphilis, approximately one thousand times as many. This comparison is disgraceful to us.

Sweden stamped out syphilis partly by making it reportable. That is, the physician and the patient were by law obliged to report every case of syphilis. Next, they made treatment compulsory. Whether the patient wanted treatment or not made no difference. Third, they made treatment easily available, both geographically and economically, to the patient. Next, by a social service scheme they checked back on all contacts. That is, when a patient came in with syphilis, they found out where he had contracted it and then went to that source and treated the source. They also found out whom he had exposed, examined them, and treated them, if necessary.

Then, in addition, there is the treatment of the syphilitic, pregnant mother. We know that syphilis in the pregnant mother is disastrous. About 25 per cent of the pregnancies are lost, 25 per cent are born dead, 25 per cent are born alive with signs of syphilis, and only 25 per cent apparently escape it to develop it later. But, if we treat the syphilitic woman all through her pregnancy, we are pretty certain of her having a normal child.

To summarize, I would say that the problem of syphilitic dental dystrophies can be solved only by stamping out syphilis in our country, by treating all syphilitic pregnant women throughout their pregnancies or, possibly, by beginning treatment in the newborn syphilitic child immediately after birth.

There is a very excellent book by Parran which I would recommend to you entitled *Shadow on the Land*. It was written for the layman, but it has such extremely interesting information in regard to the public health approach to syphilis that I think we should all read it.

# THE ROLE OF ALLERGY IN ORTHODONTIC FAILURES

HENRY STAFFORD, M.D., OAKLAND, CALIF.*

AS I understand it, the object of this discussion is to examine critically some of the constitutional factors which prolong orthodontic care and at times lead to an unsatisfactory result.

No medical or dental specialty is in itself sufficient. When the orthopedist sets a fractured hip, in order for him to predict the rapidity or completeness of healing with any certainty, it is often necessary for him to call upon the clinical laboratory or a roentgenologist and so determine evidence of his patient's mineral metabolism. By the same token, the orthodontist cannot be sure of a prompt realignment of teeth or know his work will be permanent without some consideration of the factors which caused the malocclusion and which by their continuation may undo part or all of what has been accomplished. Allergy is one of the constitutional factors which may inhibit or prevent the satisfactory solution of an orthodontic problem.

Asthma, hay fever, eczema, and hives are considered as manifestations of an unusual reaction to ingested food, to inhalants or pollens, even by those who believe that allergy plays only a minor role in clinical medicine. Certain other symptoms, as abdominal pain, migraine, renal colic, and pylorospasm are thought of as allergic phenomena only by those enthusiasts who have brought allergy to its proper place in medicine. But these evident symptoms of the allergic state do not concern us here. The more fundamental manifestation, and the one which concerns the orthodontist, is the interference with normal growth pattern through food and, less often, inhalant sensitization. As in infectious diseases, so in allergy, it is the continuous activity of a subclinical condition which results in the greatest and most permanent harm.

At birth, each human organism starts with certain individual possibilities of mental and physical growth, an ultimate beyond which it cannot go. Providing that good health persists, growth will continue in a regular manner without interruption, and the potentialities of the individual will be realized. Any deviation from the so-called normal during the developmental period affects the growth pattern.

The late T. Wingate Todd of Cleveland demonstrated transverse scoring at the ends of some of the long bones which he believed were visual evidences of the slowing down of the growth process. He reasoned that in health mineral arrives at the growing end of a bone at a constant rate and is laid down at a constant rate. But when there is some disturbance, capable of interfering with growth, the mineral is laid down over a smaller area in a sufficient amount to cast a shadow on the x-ray plate. These fine lines which do not reach completely across the shaft of the bone and which disappear within two years were attributed in certain children to the effect of active food allergy in the growing child. Todd and his co-workers even went further. They felt it was possible

* Chief of the Department of Pediatrics, Alameda County Hospital.

by measurements to determine the duration of the allergic phases and even to distinguish between the lines due to allergy and those resulting from an infectious disease.

Some of the deductions of Todd from his studies have been questioned. They have served, however, to re-emphasize the interference with the growth pattern so evident in the allergic child. The physical makeup of the individual who has come in contact with some foreign substance, to which he was sensitive early enough in life to leave its mark, is typical. He is thin, short for his age, and below the average in weight for his height. His musculature is flabby. His face is flat and narrow. There are granulations on the outer half of his conjunctiva. His nasal mucous membrane is congested. He tends to rub his nose intermittently or make rabbit faces. His palate is high, arched, and narrow. Mentally he is irritable and cries easily. He can keep his attention focused for only a short period of time. He is hyperactive and then suddenly listless; both evidences of extreme fatigue. Physicians who have followed children from birth through puberty see many such children. And I have seen them in the offices of orthodontists and heard the complaint that the correction of their malocclusion was at a standstill.

We can go further and more carefully examine the facial contours of such a child to determine the anterior, lateral, and vertical growth. Dr. Milton Cohen demonstrated the following points to me in a markedly allergic child. If you run your index fingers downward over the malar prominences from the center of the orbit on both sides, it can be determined whether these structures are normally rounded forward, or whether the face is flat, or even concave, in these areas. This is a measure of forward growth. Continue the index fingers downward and press toward the midline, just below the malar prominences. You will find the approximate width of the jaw. This is a measure of lateral expansion. With the child's mouth open, the disturbance in vertical growth is evidenced by the curving upward of the anterior end of the hard palate, and at times by the forward and upward inclination of the maxillary incisor teeth. Cohen believes that "allergic children with marked disturbance in facial growth, particularly in lateral expansion, have had more or less continuous allergy since its onset during the first year of life. Those in whom there is found good lateral expansion with only moderate flatness due to disturbances in anterior growth usually give histories which date back only to the second or third years."

Now, you might well remark that the allergist is only a symptom treater. That to some extent is true. By means of skin tests he finds that an individual gives reactions to certain articles of food, inhalants, and pollens. With the clues furnished by the positive reactions to foods, aided by elimination diets, symptom-producing foods are removed from the diet. On the basis of pollen and inhalant reactions, desensitization is instituted and the resultant symptoms diminished or completely controlled. But the patient remains an allergic individual. If he comes in contact with any of the antigens to which he is sensitive, any or all of his previous symptoms may recur. Nothing has been done to alter his underlying allergic pattern.

But it must be remembered that the allergic adult, symptom free, can lead a normal, active life; if untreated he is often unable to do so, or, at best, he is able to carry on with diminished efficiency. Even more is accomplished

in the growing child. Separated from food to which he is sensitive and desensitized to inhalants and pollens, his potentialities of growth can be realized. Moreover, both the adult and growing child have less chance of future reactions the longer they remain symptom free.

You might further ask, "If a child remains symptom free, can one be certain that normal growth is being maintained?" For the average clinician it is impossible to follow growth in relation to symptoms such as wheezing, sneezing, or hives, in the elaborate manner practiced at the Brush Clinic in Cleveland. There, x-ray studies are made at frequent intervals to determine faulty mineralization and the appearance of scoring in the long bones as an index of growth. But, in a symptom-free child, average increment in height and weight plus normal psychologic reactions do serve as a very fair yardstick of growth in general, and for our purposes, growth of the skull in particular.

In order to illustrate, let us take one concrete example of an orthodontic problem in an allergic child. A boy, aged 11 years, 9 months, was first seen on March 29, 1934, complaining of full summer hay fever, with nasal stuffiness in the winter months. His allergic difficulties began at three weeks when difficulty in breathing was noted. Colic began at seven weeks. Eczema started at one year and continued until the third year. There were frequent colds from the first year and, from the seventh year on, definite hay fever appeared. Orthodontic management of a malocclusion was continuous from 1930 to 1933, but little had been gained as there was failure of good bone formation in the jaws. This was due to poor mineral reserve in the bones as evidenced in x-rays of the hands.

Studies from the allergic standpoint proved him sensitive to grass pollen, ragweed pollen, and house dust, and suitable management was carried out over three years' time. The symptoms became progressively less marked, mineral reserves increased, a good result was obtained by the orthodontist, and he was discharged in September, 1937.

This is a condensed case report from the files of Dr. Milton Cohen. It was selected because the Cleveland group, with whom Dr. Cohen has been associated, has been thinking of allergy in relation to growth for a sufficient time to have passed the impression stage.

Now, what inferences can we draw? Uninterrupted, growth is a continuous process which proceeds in a regular manner. Allergy is one of the many factors which can retard normal growth. The bones which concern the orthodontist share with other bodily structures which fail to follow the normal developmental pattern of a given individual. There are certain physical attributes which characterize the allergic child that are easily recognized. In many children, if the allergy remains untreated, certain types of malocclusions may result, orthodontic correction can be unnecessarily prolonged and partial and at times complete failure may follow.

There is only one solution; the orthodontist must learn to suspect, if not definitely recognize, allergy in childhood and there must be a closer cooperation between the physician genuinely interested in allergy and the orthodontist. It is another example of the need of a more closely unified dentomedical team.

2940 Summit Street

# DUCTLESS GLANDS

H. Clare Shepardson, M.D., San Francisco, Calif.

DR. MARSHALL has pointed out the pathologic changes that happen in bone. Now, certainly, there is a very definite relation between the function of at least some of the ductless glands, and the development that occurs in the bony structures of the body. It follows, then, of course, that if there is a marked disturbance in those bony structures with which development of the teeth is parallel, the work the orthodontist does is apt to be strewn with difficulty, if not with failure.

There are certain of these abnormal glandular conditions that occur in childhood which can be very readily recognized. I have selected three or four cases which will illustrate the utter hopelessness of attempting to practice orthodontic work on individuals who are afflicted with these particular conditions. These are the more common endocrinopathies and the ones that may be recognized by superficial examination.

The first case is that of a child who represents the effect of a lack of thyroid function, the so-called juvenile or childhood myxedema, which is frequently erroneously termed "cretinism," though true cretinism we do not have in this country. This child would appear and did appear to be a child of one year of age. Actually the child was 12 and was of necessity carried because he was unable to walk.

The typical case of juvenile myxedema is very obvious. The child is somewhat obese. He is mentally retarded and markedly dwarfed in stature. The skin is thick and dry. The hair is sparse, coarse, and dry. The face is broad and round, with a low wrinkled forehead; a rather low hair attachment on the head is the rule. The eyes are sunken and wide apart. The lid slits are narrow and the lids themselves puffy. The "saddle" nose has some resemblance to the nose that Dr. Templeton referred to which occurs in congenital syphilis and in achondroplasia, which is another type of dwarfism. The tongue is large, thick, and juicy and often protrudes so that the mouth gapes open and drools. The combination forms a face that is at once stupid and repulsive.

Of more interest to you, the condition of the teeth depends upon the age of onset of the disease. Marked delay in eruption of both the temporary and permanent teeth is almost a constant finding.

This child, when x-rays of the bony development were taken, had what is termed a "bone age" (which represents the degree of nuclear development of the bones) of one year. It had a mentality of about one year. And I am quite sure that any of you will agree that the most meticulous orthodontic work would not have helped this child's teeth until the glandular condition had been in part at least, remedied.

Associate Clinical Professor of Medicine, University of California Medical School, San Francisco, Calif.

And therein lies another factor. Administration of thyroid to this child resulted in a phenomenal growth. I think it was eleven inches in two years (normal growth would be four inches). There was also a very rapid development of the face and jaw and rapid eruption of the teeth. Now, to be sure, I do not expect that any of you would attempt orthodontic work on a child of this character, but there are many children who have thyroid deficiency, perhaps not to the marked degree that this child has, but who in general resemble this child to a greater or lesser extent. Treatment in these latter cases, just as in the more severe types of juvenile hypothyroidism, results in rapid changes not only in body contour but in the dental formula as well.

There is one condition which, unfortunately, is frequently confused with this and for which we are unable to explain the etiology; that is the so-called Mongolian idiot. But Mongolian idiots do not have a retarded bone age or, if it is retarded, it is retarded only very slightly. The almond-shaped slanting eyes and the incurvation of the fifth fingers help to differentiate this congenital disease.

I think also that orthodontic work in a Mongolian idiot would be a useless procedure!

The second case is that of another type of dwarf. The pituitary gland secretes a material which stimulates growth, and, if there is a deficiency of this material, there follows a failure of growth. This failure affects the bones, but only insofar as *growth* is concerned and not insofar as *development* is concerned. In other words, the bones develop but they do not grow.

We have as a result the so-called hypophyseal dwarf. The bone age of this child is apt to be normal or only very slightly retarded, but I am quite sure that none of you would confuse a child of this character with the one that just preceded.

The skeleton of the hypophyseal dwarf is invariably delicate and gracile as compared with the clumsy, thick appearance of the hypothyroid dwarf. And the palatal arch is narrow and very highly vaulted. The teeth are apt to be overcrowded and forced out of alignment by reason of the inadequate development of the jaws. The child's mentality is usually normal and I think that even without taking the child's clothes off, which I believe you rarely do, you probably would suspect the disturbance from the facial appearance, the slightness of build, the mentality of the child and the formation of the mouth.

Again, treatment of this condition results in development, and with it would develop the jaws and the teeth. Untreated cases would be very unlikely to respond properly to orthodontic procedures, whereas treated cases might well overrespond. And it seems to me that the logical course to follow would be to delay such orthodontic procedure until glandular treatment has been instituted and beneficial effects obtained.

The other endocrine organs that have to do with growth are the gonads. However, gonadal deficiency does not produce dwarfism. On the contrary it produces to all intents and purposes gigantism. Yet, here, again, diminution in function results in a marked failure of bony development. The bone age is retarded. There is an adequate amount of growth hormone secreted by the

anterior pituitary gland but the eyiphyses are not closed so that the bone continues to grow rapidly. The result is a tall, slender child.

The third case illustrates the ill effects that are produced by a failure of gonadal function in childhood or prior to adolescence. The preadolescent eunuchoid is tall, slender, undernourished, with a narrow chest, with long, slender fingers and long, narrow feet. Here, again, the palatal arch is high and vaulted, and the teeth are crowded. And here, too, treatment of the condition, particularly with the newer products that we have available at the present time (in contrast to those impotent materials of a few years ago) results in a marked improvement in the bone age as well as the other factors in the child's development. As a result of treatment this preadolescent eunuchoid boy's body configuration has changed. Even his face has changed. Certainly it was obvious in the boy himself. And if this change had been produced at an early age when orthodontic work might have been done (I do not believe there was any done on this particular patient), I am sure that, unless treatment of his glandular condition had at least paralleled the orthodontic treatment, your work would have been doomed to failure and you to disappointment.

These are endocrine disturbances which markedly affect bony development and development of teeth, as well, by retarding them. There are some interesting types of glandular disease that result in very profound advancement in bone age and in development. The next case is that of a boy, 5 years of age. He is not a dwarf, although eventually he will be a short individual. He is really a giant at the present time. He has a bone age of 12 years and the tooth development of 12 years of age. Development in the other parts of his body is even more advanced than that.

This boy had a tumor of the adrenal cortex. Incidentally, he is alive and healthy today, he being one of nine cases which have been reported by my associate, Dr. H. Lisser, in medical literature and the only one, as far as I know, that has been successfully operated upon.

These tumors are most frequently malignant, but their glandular disturbances occur before the malignant cells tend to break through the surrounding capsule. A tumor about the size of an apple was removed. This boy had a moustache, and a very worried, tense look. I am sure that if somebody had told you that he was five years of age you would have immediately suspected that there was something wrong.

There are other ductless glands that produce disturbances of this character, and we occasionally see them in individuals who, as far as we know, have no ductless gland disturbance at all.

There are just one or two additional things that I wish to mention briefly.

Going back to the case of the hypophyseal dwarf—all of the disturbances which occur and which produce dwarfism (and which are not to be confused with the previous case, the hypothyroid dwarf) are not due to a pituitary deficiency. They may be due to dietary disturbances. These are the types of patients to whom Dr. Stafford referred, in part, at least, and you can see wherein they conform to his description.

Prior to the time we had insulin to treat juvenile diabetes properly we saw the so-called diabetic dwarf which looked quite similar to this child (the hypophyseal dwarf). The retardation in growth was due to improper nutrition of the child, which in turn resulted from actual failure to metabolize that food which may have been ingested. We rarely see this type of dwarfism now that insulin and especially protamine zinc insulin is available, although we do see other disturbances which are the result of juvenile diabetes.

Another condition which is far more common than the dwarfism resulting from juvenile diabetes, but which is still relatively uncommon, is the so-called renal dwarf or the dwarfism of renal rickets. A renal dwarf looks quite similar to this hypophyseal dwarf and a differential diagnosis, even by many physicians, might well be difficult because their bony structure is quite similar and as far as you are concerned, the construction in their mouth is quite similar. They, however, have a very profound anemia, a marked reduction in their phthalein output and a marked elevation of the nitrogenous elements in the blood. The prognosis is not very good. And consequently, I would suggest that you not attempt orthodontic procedures on them since your results are certain to be disappointing. Usually you will not have a sufficient amount of time to do very much.

This, then, gives a brief outline of some of the effects disturbance in function of the ductless glands can produce. Some of these effects are very profound and some very slight. Like Dr. Stafford, I think that much can be gained from cooperation between the orthodontist and the clinician who is interested in the child's specific trouble. It may be a glandular disturbance, it may be a luetic disturbance, or it may be allergic or a disturbance resulting from malnutrition. But irrespective of the etiology, until those causative factors are corrected, I am sure that you will agree that your orthodontic problem will continue to be extremely difficult, if not impossible, to solve.

# THE INTERNIST'S CONTRIBUTION TO THE ORTHODONTIST

S. P. Lucia, M.D., San Francisco, Calif.

PERHAPS my remarks will come as an anti-climax after you have heard the able discussions of Doctors Templeton, Stafford, and Shepardson. May I tell you that the contributions in the fields of dermatology, pediatrics, and endocrinology are part of the internist's contribution to the practice of orthodontia. Doctor Templeton has discussed with you the effect of the growth-inhibiting processes of infectious origin which operate during a period of great and rapid bone growth. Fortunately he has used the disease, syphilis, as an outstanding example of this phenomenon. Doctor Stafford, in referring to the work of Todd, brought into high relief the discussion and significance of "lines of arrested bone growth." Truly here in bone is writ the health history of the patient. These "lines" are as telltale in a way as the fragments which the archeologist studies when he reconstructs the histories of past civilizations. One might ask of Doctor Stafford, does the allergic state, of itself, interfere with growth, or does it influence growth by altering the mineral metabolism of the host? Certainly the "altered reactivity" of the cells in allergy means that often many essential and necessary food substances must be deleted from the diet and in the growing child this may prove disastrous.

Doctor Shepardson discussed the endocrines and demonstrated how important these regulators of growth and function may be in the regulation, not only of complex physiologic processes going on within the soft tissues, but also of the quantitative and qualitative processes of growth going on in the skeletal tissues. The endocrines in many of the patients whom Doctor Shepardson discussed had gone to sleep, so to speak, and the "governors of growth were out on vacation." So you see that the three preceding speakers have adequately covered the field of the internist; that is, they discussed the effect of infections, nutrition, and the endocrines on growth. It is my belief that these three factors are most important for purposes of our discussion. Their interrelationships are marked and dramatic in the child, but they are also operating, perhaps more subtly, in the adult. Although there is little of direct benefit to the orthodontist coming from the internist, dealing as he does in the problems of adult medicine, much may be learned of the mechanisms by which changes occur which interfere with proper bone formation and function of the delicate structures of the oral cavity. The knowledge by which these physiologic processes come about may be of great value to the orthodontist, because I believe that the orthodontist, like the good internist, is practicing a form of preventive medicine.

The mouth is the "diagnostic mirror." As a child I remember displaying my tongue to the doctor and I wonder what he saw there. Today I ask my

Associate Professor of Medicine, University of California Medical School, San Francisco, Calif.

patients to do similarly for me. The story of a person's hygiene, nutrition, habits, and on occasion endocrine function is there written in the color, texture, structure and alignment of the oral cavity and its specialized structures. Furthermore the diseases his body has experienced leave their marks in the transverse ridges of his teeth, and again like archeologists, we build a hypothesis concerning the past health experiences of our subjects. By careful and thoughtful contemplation, one may be able to appreciate the subtle effects of the "termites of disease." May I state at this point that we must differentiate between the subjective symptoms and the objective signs. Surely our greatest work may be done before signs develop, and for this reason I would stress the proper appreciation and evaluation of symptoms as obtained from a painstaking history. Granted that it involves the expenditure of considerable time, but if one wishes to do anything by way of prevention, he must work to alleviate and eradicate symptoms long before signs are produced.

I shall limit my discussion to the significance of jaw pain. Very often adults complain of pain in the jaw bones. On analysis it may be revealed to the trained person that the bone demonstrates rarefaction, and that its trabecular structure is aberrant. Often the roentgenologist reports, "bone within normal limits." Fortunately as specialists you do not pay much attention to such conclusions. You are interested only in a technically perfect roentgenogram. Not so long ago a man of 58 years came to the University Hospital and gave the following story: Many months previously he had seen a dentist and had some teeth removed and some scaling done. At that time he complained of "sore jaw bones." When the dental work had been completed, he still complained of a very sore jaw and characterized it as "soreness deep down," and he pointed over both the maxilla and mandible. Shortly thereafter he consulted a surgeon and an appendectomy was done because of a combination of symptoms—nausea and pain in the lower abdomen (this was later demonstrated to arise from both ilia), and some disturbances in gastrointestinal function. While he was recuperating following surgery, he got out of bed and heard a "crack." This proved to be a pathologic fracture of the right femur. Then he saw an orthopedist and the diagnosis of Paget's disease was made. Although Paget's disease does involve the calvarium frequently, it rarely if ever manifests itself by frank lesions in the maxilla and mandible. A peculiar characteristic of Paget's disease is that the bones may be painful long before structural changes may be demonstrated in them roentgenographically. The end result of the Paget's process is aberrant bone structure, characterized by multiple areas of decalcification and disorderly attempts at repair, such that there will be areas of hypercalcification with a general distortion of the form of the bone and an increase in the temperature of the structures around and over the bone. In studying this patient carefully, we found out that during the past eight to ten years he had had occasional periods of urinary frequency. At the termination of these periods he would pass a great deal of very milky urine. The intern recorded that story, but did not attach any significance to it. This patient remained in the University Hospital for many months during which time he had three bouts similar to those noted above. The milky urine proved to be due to a flood of phosphates; in other words this patient had cyclical and

recurrent phosphaturia, following which he complained of generalized pain in the bones affected by the Paget process. We know that an excess of phosphates carried in the blood stream will irritate the kidney, producing urinary frequency and eventually a shower of phosphatic crystals. Occasionally these phosphates precipitate in the delicate tubules and produce concretions which occlude them. I mention this phenomenon because it is a prelude to many of the conditions characterized by decalcification, among them being osteomalacia, Paget's disease, and disturbances of the thyroid gland function (either primary or secondary). This patient subsequently had a second fracture in the opposite femur. Today, three and a half years after the original fracture, he is able to get along fairly well. His treatment consists of a diet rich in mineral salts with supplementary dicalcium phosphate and vitamin B.

The second case is that of a woman, 48, who consulted the dentist because she had pain in the jaw. The roentgenograms of the teeth demonstrate aberrant trabecular structure in the maxilla and mandible. Following this, x-ray films of her ribs and other long bones were made. Again the roentgenologist reported "normal bones." This patient demonstrated a low basal metabolic rate (minus 22 per cent). For many years she had tried to keep herself slim (all others in her family had been quite robust). A careful dietary history revealed that she had not taken the foods she needed and her bones showed it. We diagnosed her case as one of dysthyroidism and malnutrition. She was given small doses of thyroid substance and a diet low in carbohydrate and high in mineral salts, supplemented with calcium, phosphorus, and vitamin D. Within a month she reported to her dentist that the pain in the jaws had disappeared. Incidentally we do not expect such miracles to happen in so short a time. It will take months before health can be restored in this woman if it can at all.

A third patient is a woman of 32, who at one time suffered from functional hyperthyroidism (her basal metabolic rate was +15 per cent and +30 per cent) and was seen by a prominent endocrinologist in town. Last spring she was seen because of disturbed thyroid function and her basal metabolic rate was –5 per cent. She complained of pain in the jaw, the significance of which at that time was not suspected, and three or four months later she stated that she had to have "a lot of dental work done," and she wondered what had happened to her beautiful teeth. This past spring she returned again, this time complaining of irritability and nervousness and mental anxiety. The pulse rate was low, the surface temperatures of her body low, and a basal metabolic rate of 10 per cent minus (a hypothyroid body and a hyperthyroid mind). She was diagnosed as suffering from dysthyroidism. She was given Lugol's solution and did fairly well, although she developed marked dermatographia and complained of diffuse bone pain. Phenobarbital in small doses was prescribed with satisfactory results. Incidentally one cannot draw conclusions from these latter two cases, but they do demonstrate the type of problems with which the internist is confronted when the presenting symptom is "pain in the bones."

The last patient was an Italian woman of 34 who came down from Chico five or six years ago and had a small tumor removed from the abdominal wall.

The pathologist's diagnosis was "desmoid tumor." Later she came to the University of California Hospital and complained of rectal bleeding. A round, hard tumor was removed from the lower part of the sigmoid bowel, and it was noted to have the same characteristics as the tumor previously removed from the abdominal wall. She was discharged to her home. About five or six months later she returned to the clinic complaining of "pain in the jaws," and pointed to the mandible and maxilla. She had pain in some of the long bones, so we took roentgenograms of many areas. These demonstrated peculiar cysts and aberrant sesamoid ossifications. The films of the teeth were remarkable in that there were demonstrated multiple accessory tooth cysts both in the maxilla and mandible. It was concluded that these were sequelae of the original desmoid tumor. I report this case to you because of the similarity in symptoms but the dissimilarity of the pathologic processes in the cases above noted. This is the essence of diagnosis.

May I conclude by repeating that the internist may help you directly in a very small way, but that indirectly he may be able to offer you suggestions and explanations for the mechanisms by which these remarkable changes take place.

# ORTHODONTICS AND GROWING UP

CLIFFORD SWEET, M.D., OAKLAND, CALIF.

ORTHODONTICS has grown up in the same manner that every organism grows up. Every human being grows up mentally and then has to grow up emotionally before he becomes an adult. And the best sign that I have known that a person has become an adult is that he becomes introspective. He begins to examine himself and questions his motives and his results. And that is exactly what this meeting this morning indicates that you people are doing. You are beginning to ask why these conditions which you are called upon to treat are present and whether or not you are meeting them in the most adequate manner.

Every specialty of which I know (and orthodontics is no exception) has a certain history. The specialty arose, in the first place, in response to a definite and crying need, a need which was not being met. And a few men, because of their intellectual curiosity, because of their anxiety to serve their fellow men, have begun this study. While the work of any specialist is perhaps somewhat difficult to sell right at the beginning, the need is so great and the obvious results accomplished so beneficial that at first he is overwhelmed with work or, as I have sometimes thought of it, the harvest being great, the workers being few, the gleanings are so abundant also that one does not have to stop to ask many questions; he has not time. But there are, fortunately, always a few great spirits. And I believe they are more prevalent among professional men who have subjected themselves to a long and arduous course of training than among any other group of human beings, who begin from the beginning to ask: Why has this condition arisen? What can we do to prevent it, and what can we do to improve it as rapidly as possible, as economically as possible, and with the fewest harmful by-effects to the patient?

There is no specialty in the treatment of human ills that stands by itself. Each is related to all the others, as our other speakers have mentioned this morning. Even obstetricians in these days are beginning to talk about some orthodontic problems that have their beginning in the birth of the child.

Dr. Templeton has spoken about syphilis before the child is born. I would like only to add to Dr. Templeton's discussion, if I may, the fact that the intrauterine treatment of the child for syphilis can produce a child free from any of the stigmas of syphilis only if it is begun prior to the fourth month of gestation. And I believe there is a very practical way in which all of us who are interested in human beings can do something, and that is to encourage the young people to have Wassermann reactions done before they are married and to encourage them to go to the obstetrician who will see that the mother has a Wassermann reaction before or early in her pregnancy. I believe that is mandatory at present in California, but it may not be in other places.

Pediatrician, Chief of Staff, Children's Hospital, Oakland, Calif.

There is some evidence to indicate that the child's jaws, and facial bones may be displaced during the progress of his head through his mother's birth canal and that some of the deformities may become permanent. Incidentally, if the child's facial bones are displaced during birth Nature has provided a magnificent remedy for putting them back into place, and that is simply to nurse his mother's breast. There we have an additional reason for encouraging mothers to breast-feed their infants.

When the child suckles at his mother's breast he does not actually suckle so much as he chews. He must bring his jaws up into perpendicular apposition in order to get milk out of his mother's breast. While, when he is fed one of these damnable, long nipples that are commonly used on a nursing bottle, he does not use his jaws at all but, seizing the nipple between his tongue and his hard palate, he simply pushes it with his tongue to get the milk out. And repeatedly we have seen a potential Andy Gump, with jaws that looked as though they would be useless to him at birth, very rapidly get an excellent position of his jaws after just a few weeks of nursing his mother's breast.

Dr. Edwards, a dentist in Oakland, has made an excellent contribution to public welfare in his invention of a nipple which has a ledge on it resting against the child's maxilla, which forces the infant to bring his mandible up while he nurses. And we have in several instances been able to prevent an orthodontic deformity by prescribing that type of nipple. In some instances, when the child was unable to nurse the normal nipple because of the deformity of his jaws, as soon as he was taught to bring his jaws up he could then nurse.

It is certain that during the process of birth the cartilage of the nose is often dislocated out of its normal notch in the superior jaw, and if this is done at the time of the infant's birth it is a very simple matter usually to put it back. If someone in going over the baby will just take care to shove the child's nose vigorously back and forth half a dozen times, that cartilage will snap back into its central position and the child will be spared later in life having one completely obstructed nostril. And the operation for the replacement of this cartilage in the hands of the most skillful otolaryngologists has so far in my experience been unsatisfactory. Even weeks after, if it cannot be put back by this simple manipulation of the nose, it can be replaced often merely by pressure from the convex side of a Mayo hemostat; a very simple procedure.

The otolaryngologist and the orthodontist have long been associated. I shall pass this phase of your relationship with only one or two remarks.

One is that the adenoid tissue does not obstruct the breathing of an infant much before one year of age, and the removal of the adenoid from a little baby under eight or ten months of age is a useless, harmful procedure. Also, the adenoid tissue once adequately removed at one year of age or later rarely returns, and the repeated scraping of the nasopharynx or the removal of traces of adenoid tissue should not be done. It is harmful, and anyone who gets a very large secondary growth of adenoid, which does occur occasionally, is usually a highly allergic individual and you had better send him to my associate, Dr. Stafford! And anyone who takes out a secondary growth of adenoid ought to at least document his "fish story" with a specimen or with a photograph taken to scale!

Besides the allergic obstruction of the nose, which, as I shall point out in a few minutes, is one of the real problems that concern the orthodontist, there is the child who in the course of a cold gets a prolonged infection of his paranasal sinuses. Mitchell and Shea of Memphis have shown very definitely that the prolonged infection of the paranasal sinuses prevents their development and growth. And I need not argue with you that if the paranasal sinuses themselves are not developing and growing in size, the bony structures which surround them and are related to them are also standing still.

Every child who gets a sinusitis should have that sinusitis adequately treated, and since rarely, if ever, the early treatment of sinusitis is surgical in nature, its treatment is often a general medical problem. Except for those few people now left who can support an orthodontist, an otolaryngologist, a pediatrician, and an allergist all at once, the treatment should perhaps be most generally in the hands of someone who is concerned with the general welfare of the patient and is capable of looking after his nutrition and his hygiene and other things as well as the best topical treatment of his sinusitis.

There are certain mechanical factors in the life of every child which have a distinct bearing upon his orthodontic problem. Every child who is considered by anyone who thinks of children at all must be thought of in terms of the three principal influences which shape his life and his well-being.

The first is his individual, his hereditary, quality.

The second is the support or lack of support which he gets from the food which is made available to him.

The third is the effect which his total environment has upon him during the rapidly changing years when he is being molded.

His heredity may take many aspects. In the first place, I seldom see a child who has good mechanics in one part of his body and poor mechanics in another. When you sum him up, he is generally at an excellent level, a good level, a fair level, a poor level, a bad level, and so on, concerning his general body mechanics.

The equipment which he gets out of his heredity may be a nose which is very narrow, very small and one which has very little capacity for the passage of air. He may have a very tiny nasopharynx. Sometimes even in a good-sized child I can barely get the tip of my finger within his nasopharynx even though at that time he has had very little deformity of his hard palate.

Then there is the child whom we see every once in a while who has had his thymus gland mercilessly x-rayed because he makes a noise when he breathes. Just for purposes of my own description I call that child one whose upper respiratory tract is not made of "good cardboard" but of "wet cardboard." The cartilaginous structure of his nose, of his nasopharynx and even of his larynx is very poor and has very little elasticity. They collapse even in the act of breathing, and if you look at certain little babies you will see that the nose actually closes with each inspiratory effort. These children make a crowing noise and they are generally a year old before they recover from this stridulous type of breathing. When one looks at them with a direct laryngoscope one can actually see the sound box, the Adam's apple, close up as the child makes an attempt to breathe. That fellow deserves a lot of help. And I would only say

in passing that I would not desert these children and leave them to their fate. I would make a particular effort to so shape that child's environment that he gets as little harm as possible done to this tender, easily injured body of his.

The way in which the child sleeps is important as a factor of his environment. Every child should sleep on a firm bed. When he sleeps on one that big brother and big sister have already slept in, one that is hollow down in the middle, he sleeps with his chest caved in. No child should have a pillow when he sleeps. When he sleeps on his back, as he very often does, with a pillow his head is thrust forward and his chest wall is depressed during all the hours that he spends in bed. If the child sleeps on his abdomen and has a pillow in bed, he still has his head in an unnatural relationship to his torso, and it interferes with the free use of his diaphragm and extrinsic muscles of respiration during sleep.

I have no objection, contrary to the opinion which is expressed by some of my orthodontic friends, to a child sleeping on his tummy if he wants to. I think it is harmless. I think not only that; it is good. Because when the child lies face down in his bed he constantly practices lifting his head and I think, as I shall explain to you as I go along a little further, that the factor with which we are most concerned so far as the mechanics of his respiration go is the full extension of the spine. You will think of the spine as I go along as though it were a rubber band and I am constantly asking you to stretch that rubber band, make it long.

I think it is a very simple thing to ask any mother who has a young baby to put the baby's head north one day and south the next. Reverse the way he lies in his bed because it is natural for the child to make certain movements toward the light and to follow his mother as she goes about the room, and if he does it from the right one day and left the next he will have an influence for symmetric development.

I think that the child's orthodontic condition is affected by the kind of shoes he wears when he begins to walk around. And you will wonder why. But I believe that to be true, because, again, I relate the mechanics of the jaws to the mechanics of the entire body.

Now, just as soon as a child begins to walk he is ordinarily put into a rigid shoe which prevents him from walking in the way which nature intended him to walk. When a baby first stands up he always stands up with his feet everted, pronated and with all of his joints in slight flexion in order to get his center of gravity as near the ground as possible and give himself a wide base. When he takes his first step he does it in this fashion. He simply plants a foot forward to gain a new base. Once he begins to gain coordination and mechanical efficiency he begins to walk. But just about that time somebody puts a shoe on him that is too short, and 75 per cent of the children that I look at who have been in my office have shoes on that are too short. And when you have a shoe that is too short you have to evert and pronate and walk like this to get the weight off your toes. Or else he has been put into a shoe that is too rigid. Somebody has noticed that he has a little physiologic pronation, and so they put a great heavy sole under his foot. Therefore, the child being forced into pronation and eversion, is also forced into a sway-back. Since we may be specialists that deal with only one part of the body we can divide it into

regions for our purposes, but Nature has forever made it indivisible, and any one part of it always affects all the others. So with his sway-back, with his rounded upper back, and with his head thrown back in order to balance himself, his spine is not in extension but has all of its curves increased. And I will ask any of you to stand in this position and attempt to breathe through the nose. You will find it extremely difficult. While, just as soon as you come up erect, your nose is free, your diaphragm is free, your bellows begin to work.

Of course, I would like to submit to any group of professional men that when they begin to talk to their patients about body mechanics it does not go over very well while the doctor stands with his feet everted and flat and his belly hung out and his head down. It was Emerson who said "What you do speaks so loudly I can't hear what you say."

Body mechanics is important, gentlemen. It is not just a passing thing that I have gone crazy about. I believe it has a profound effect upon the health of the human organism, and I believe it represents one of the very important preventive phases of pediatrics that we begin to teach people to make good use of their bodies. And when you go home tonight or, if I have interested you at all, if you will try this for yourself; with the upper back curved, the shoulder girdle depressed, it is impossible to breathe freely. If I breathe through my nose with my chest collapsed I use only the upper part of my lungs, no more. And when I cannot get air enough I begin to call on my accessory powers of taking air in, and that means that I begin to use my mouth when my nose does not serve adequately. And, of course, that is what the child does.

It is a law of Nature, to which I know no exception, that development follows only in the line of function. It is not only Wolff's law for bone. Bone is an elastic structure which can be changed at any time in life. And when one of my orthopedic friends tells someone who is 25 years old or 50 years old that it is too late to improve the function of some part of his body, I am always a little impatient about it because bone is an elastic structure that can be changed until death comes.

The only way that a child, who happens congenitally to have gotten narrow, poorly formed breathing apparatus, can improve it is to use it. And somebody has to teach him how to use it. I saw a child just yesterday afternoon with a malocclusion. His mother is taking him shortly to the orthodontist. Everything in his body is "malocclueded." His feet are turned out, his posterior leg muscles are short, he has a round upper back. Correcting his teeth is not going to correct his body, and correcting his body will not correct his teeth. That boy needs the help of a skillful orthodontist. But at the same time, unless somebody helps him, he is going to go on being a mouth-breather even after his malocclusion is corrected, and he is going to walk in the typical Mr. and Mrs. America fashion down the street. The next time you are up in your office five or six stories or in any place above the street, just look down at the people walking past and see if I have exaggerated what they are doing.

It is important that we think of the entire child, not merely of just any part of him. These children can be made over very simply. I hope some day we will get the physical education departments of the schools to wake up

to their responsibilities and begin to do something for these children instead of leaving them, as they do, going on year after year and year after year getting worse and worse when they could so easily be made better.

Even the child's chair is important. The child graduates from his high chair and is put into a chair that was made for an adult. He sits at the table, and I go into a house and I see him fishing around with his feet trying to find some place to rest them, and his father saying, "Sit up there! Why don't you sit up!"

If I sit on anything that is too high for me, the weight of my own legs throws my shoulder girdle down, and when my shoulder girdle is down I cannot breathe. When I sit in a chair with my feet planted firmly on the floor I can sit up. And so, if the poor child merely had a footstool for his feet while he sits at the family table, there would not be nearly so much squabble, and the child would not have his shoulder girdle dropping down so much of the time as he does now.

I want to leave with you this word: that the child who has an orthodontic deformity generally has a deformity that extends throughout his whole body. And I am not going to stop right now without leaving another thought with you.

I spoke of the early days of specialties. In those days, if the child went to the orthodontist, he did not go to much of anybody else and there was not such a burden put upon the family purse as there is now. So often I see a child whose mother tells me that she cannot afford to have any kind of medical care for him because all of the family funds are going for orthodontics. And then I meet one who is having orthodontics and going along with it all right, but some member of the family falls into the hands of the surgeon and then things are all upset. Then there is somebody who gets pneumonia and they have to have a pediatrist, and there are just too many people to pay for out of that family budget to manage.

I think that our fundamental problem in this day of specialties, in this day of trying to do something constructive for the child, ought somehow to include an agency to which these people can go and have a fair appraisal of their ability to care for their children and a fair distribution of the amount that they are able to pay among the people who can and will do the work.

I know this is a revolutionary thought. I know all about human selfishness. But in such groups as this I know there is a large element of altruism and I know that our underlying purpose is to do more and more for people for less and less, not less and less for more and more.

There must be in the near future, if we are going to meet the situation of medical care for children, at least some way in which the honest, decent people with a reasonably good income can meet their problems. You know, most of the people who have young children are also in the young stage of their income. They may have plenty ten or fifteen years from now, but it is too late to do the things that the child needs.

If we are going to meet this problem, all of us who are interested in the care of children must find a way to meet it collectively. We must give to certain of these children the advantage of a team that can meet their dental and medical needs on a basis which decent, honest working people can pay for.

# Original Articles

## STUDIES IN TOOTH DEVELOPMENT: THEORIES OF ERUPTION

MAURY MASSLER, D.D.S., M.S., AND I. SCHOUR, D.D.S., PH.D., CHICAGO, ILL.

### INTRODUCTION

ERUPTION is one of the four processes concerned in the development of the teeth. A tooth may have successfully passed through the first two stages of development (growth and calcification) and yet be functionally useless if the eruptive process is disturbed (Fig. 1). Eruption is therefore a very essential phase in the sum total of tooth development, and yet less investigative work has been done to elucidate the basic mechanisms involved in this process than in the other developmental processes. In recent years, researches on the growth and calcification of the teeth have almost completely overshadowed the experimental work on eruption.

While growth and calcification are processes that occur in other parts of the body, eruption is peculiarly a dental problem. The eruption of teeth is of particular interest to the pedodontist and orthodontist, but there are many aspects to the problem that are of immediate interest to every dental practitioner and to the pediatrician.

*Definition of Eruption.*—Before beginning our discussion, it is well to attempt to define what we mean by the term "eruption." Eruption is here used to designate the process whereby the forming tooth migrates from its intraosseous location in the jaw to its functional position within the oral cavity. Thus it involves much more than the "cutting" of the tooth through the gum tissue. Noyes, Schour, and Noyes[23] describe six anatomic stages in the eruption of the teeth (see Fig. 1):

Stage I: Preparatory stage (opening of the bony crypt)
Stage II: Migration of the tooth toward the oral epithelium
Stage III: Emergence of crown tip into the oral cavity (beginning of clinical eruption)
Stage IV: First occlusal contact
Stage V: Full occlusal contact
Stage VI: Continuous eruption

*Review of Literature.*—Many theories have been advanced to explain the mechanism which causes the movement of the tooth during eruption. They naturally revolve about the various changing histologic structures and the processes that characterize tooth development. These theories and a short summary of their essential character are given in Tables I and IA. The mechanism of each theory and the implications as advanced by its proponents will be discussed in greater detail during its evaluation. Tomes,[32] Constant,[7] Eichler[9] (Table IA), Demolis,[8] Weber,[33] Brodie,[3] and Reichborn-Kjennerud[26] give sum-

From the Department of Histology, College of Dentistry, University of Illinois.

maries of some of the prevailing theories of eruption and some of the evidence for and against them.

*Purpose of Paper.*—The large number of theories advanced to explain the mechanism of the eruption of teeth indicates that we know very little about the process. It is our purpose first to test the validity of each theory of eruption by the results of clinical and experimental findings; second, to ferret out those facts about eruption which could withstand this scrutiny; third, to integrate these facts with the hope of effecting a better understanding o the mechanism of eruption; and last, to suggest, on this basis, further experimental studies on the problem.

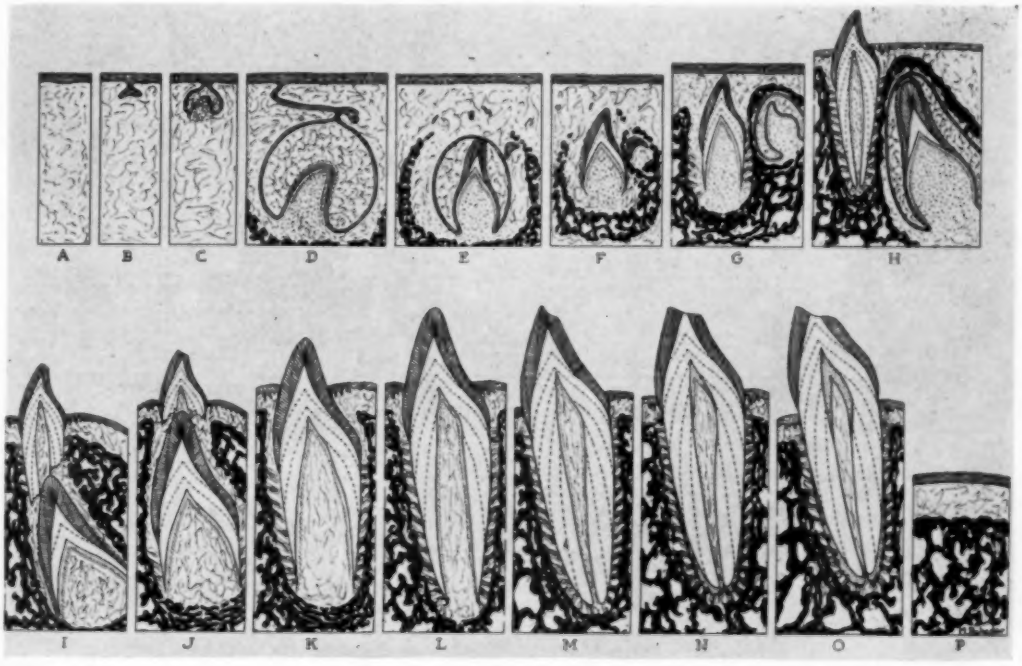


Fig. 1.—Diagram illustrating the stages in the development of the tooth. Life history of the deciduous and permanent dentitions.

## METHODS AND MATERIALS

Our first approach was to test each theory in the light of the clinical and experimental evidence for and against its thesis. The clinical data used in this study were obtained from the literature and from clinicians who have kindly given us the results of their many years of experience.

The experimental data were obtained mainly from investigations that have been conducted in this laboratory since 1932 on the effects of various endocrine, dietary, and mechanical factors on tooth development.

One of the prime requisites of an experimental study is the use of a suitable experimental animal. For this purpose, the rat with its continuously growing and erupting incisors is useful. Most of the experimental studies were therefore conducted on this animal.

It is seldom that the results from a given experiment cannot be re-evaluated and interpreted differently and yet logically, depending upon the different ob-

TABLE I

THEORIES OF ERUPTION

| | THEORIES OF MECHANISM OF ERUPTION | PROPONENTS | EXPLANATION |
|---|---|---|---|
| GROWTH OF DENTAL TISSUES | 1. Growth of root | Hunter<br>Magitot<br>Nasmyth<br>Koelliker<br>Sarazin<br>Wedl | Elongation of root pushes crown into oral cavity |
| | 2. Growth of dentine and pulpal constriction | Zuckerkandl<br>Wallisch<br>Walkhoff<br>Eichler<br>Eidmann | Pressure from growth of dentine and constricting pulp forces tooth into oral cavity |
| | 3. Growth of periodontal tissues: | | |
| | A. Growth of and pull by periodontal membrane | Underwood<br>Landsberger | Movement of soft tissues surrounding tooth pulls the latter into oral cavity |
| | B. Growth of alveolar bone | Brash<br>Nessel<br>Hermann | Growth of alveolar bone carries or pushes the tooth into the oral cavity |
| MISCELLANEOUS THEORIES | 4. Pressure from muscular action on alveolar process | Berten | Pressure from cheek and tongue musculature contracts the alveolar process and squeezes the tooth into the oral cavity |
| | 5. Resorption of alveolar bone | Aichel<br>Weidenreich | Resorption of alveolar crest exposes tooth into oral cavity |
| TISSUE TENSION THEORIES | 6. Pressure from cellular proliferation | Eidmann<br>Aichel<br>Oehrleins<br>Zuckerkandl<br>von Korff | Osmotic pressure or tissue tension resulting from (1) proliferation of cells, (2) vascular bed, or (3) both, in the pulp and periapical tissues pushes tooth into oral and cavity, the roof of the bony crypt being resorbed by pressure atrophy |
| | 7. Pressure from vascular bed in<br>A. Pulp<br>B. Periapical tissues | Constant<br>Leist<br>Fischer<br>Mahé<br>King<br>Baume (medullary theory) | |

TABLE IA

EICHLER'S SUMMARY OF THEORIES OF ERUPTION*

| | | |
|---|---|---|
| MECHANICAL FORCES | Forces arising from within the tooth | Root dentine growth (Wedl, Calembrum-Mercure)<br>Root pulp growth (Hunter, Zuckerkandl, Eichler, Wallisch, Walkhoff, Kallhardt) |
| | Forces arising outside the tooth | Granulations at the alveolar floor (Baume)<br>Contractibility of the jawbone (Nessel, Albrecht)<br>Muscle pressure (Berten)<br>Mastication pressure (Albrecht, Robin) |
| PHYSIOLOGIC FORCES (Adloff) | | |

*From Scheff's Handbuch der Zahnheilkunde, 1922, Wien, Hölder, Pichler, Tempsky, p. 533.

jectives, viewpoints, and background of the investigator. The results obtained from previous experimental studies were therefore restudied and re-evaluated in relation to the problem of eruption. When no previous experimental data were

available, or when the results of other experiments were inadequate or inconclusive, new experiments were devised to test directly the validity of a given theory. Some of these experiments were reported separately by Herzberg and Schour.[12]

A summary of the clinical and experimental data utilized in this study is presented in Tables II and III.

## EVALUATION OF THEORIES OF THE MECHANISM OF ERUPTION

### 1. ROOT ELONGATION

The simplest and most obvious mechanism of eruption would be that the crowns of the teeth are pushed into the oral cavity by virtue of the growth and elongation of their roots (Hunter, Magitot, Nasmyth, Kolliker, Sarozin, Wedel, cited from Weber[33]). Tomes[32] and others point out the inadequacy of this theory to explain many of the phenomena associated with normal and abnormal eruption.*

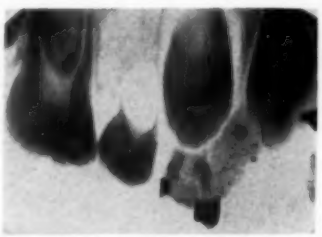

Fig. 2.—Intraoral roentgenogram of a permanent upper second premolar literally "jumping" into occlusion due to the premature loss of its deciduous predecessor. Note the lack of root formation; the dentine growth and pulpal constriction; the failure of the alveolar bone to keep up with the tooth; and the wide periapical space between tooth and bone. Compare with the first premolar which normally precedes the second in its eruption.

*Clinical Evaluation:*

*Evidence for Theory.*—Root of tooth elongates as crown erupts into the oral cavity. This evidence, however, is only circumstantial.

*Evidence Against Theory:* 1. Rootless teeth often erupt without the concomitant elongation of the root. This condition can be seen in (1) the erupted crowns of incisors or premolar teeth after the premature extraction of their deciduous predecessors (Fig. 2)†; (2) cases of congenital lack of root formation (Figs. 3 and 4); (3) the occasionally erupted but rootless incisor in the newborn infant; and (4) the eruption of the incisal segment of a tooth after fracture (Figs. 13 and 14).

2. Submerged teeth often continue the formation of their roots but do not erupt. After a long period of years and after root formation is completed, they may suddenly erupt.

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*Orban[25] has indicated that Hertwig's epithelial sheath remains at a relatively fixed point within the jaw during the eruption of the tooth. This might indicate that eruption results from root elongation or the proliferation of Hertwig's epithelial sheath pushing the crown occlusally. In a personal communication, Orban indicates that such a relation is not causal but probably coincidental.

†It sometimes happens that a bicuspid tooth will not erupt for some time after the extraction of its deciduous predecessor. Its eruption may in fact be markedly retarded. In such cases it is found that the soft tissue of the bone overlying the tooth has become markedly fibrotic and dense, and offers a mechanical obstruction to the erupting tooth. For these reasons, eruption of a bicuspid may sometimes be delayed instead of accelerated following the extraction of a deciduous tooth.

TABLE II

CLINICAL EVALUATION OF THEORIES OF ERUPTION LISTED IN TABLE I

| | CLINICAL PROCEDURES OR OBSERVATIONS | FINDINGS | CONCLUSIONS |
|---|---|---|---|
| 1. | a. Eruption of rootless teeth<br>b. Eruption of teeth after root formation is completed.<br>c. Cuspid and other teeth erupt a distance greater than total length of its root<br>d. After the premature extraction of a deciduous molar, the premolar erupts rapidly although the root remains unformed<br>e. Total length of root is attained after clinical eruption is completed<br>f. Root does not reach bottom of alveolus and therefore cannot push against it<br>g. Changes in inclination cannot be caused by root elongation | | Eruption of tooth not related to elongation of root |
| 2. | Removal of pulp in partially erupted teeth | Eruption and supra-eruption continue | Eruption not due to pulpal constriction |
| 3B. | a. Premature extraction of deciduous molar | Bicuspid jumps into occlusion; alveolar crest and root show very little growth | Growth of alveolar bone dependent upon eruption of tooth |
| | b. Extraction of tooth | Alveolar bone resorbs | Alveolar bone cannot push upon the teeth |
| | c. Extraction prior to eruption | Alveolar bone does not grow | Eruption of tooth can occur in absence of growth of alveolar bone |
| | d. Tooth does not touch walls of alveolus<br>e. Multirooted tooth could not be squeezed out of alveolus<br>f. Teeth in dermoid cysts often erupt in absence of bony base | | |
| 4. | Teeth erupting lingual to arch are not subjected to muscular action | | Eruption not due to muscular pressure on alveolar process |
| 5. | Alveolar process *increases* in size during eruption of teeth | | Eruption not due to resorption of alveolar crest |
| 3A & 6. | Clinical data lacking | | |
| 7A. | a. Pulpitis (hyperemia of pulp) | Tooth does not elongate or supra-erupt | Eruption apparently not related to vascularity of pulp |
| | b. Extraction of antagonist or opening bite with bite plane | Teeth supra-erupt long after pulp canal is almost closed | |
| 7B. | a. Submerged teeth under dentures | Teeth erupt under influence of hyperemia induced by dentures | Eruption of teeth apparently related to vascularity of periodontal tissues |
| | b. Submerged dentures or single teeth; operate one side, rub opposite | Teeth rubbed erupt as soon as or sooner than operated teeth | |
| | c. Periodontitis | Hyperemia causes supra-eruption or elongation of tooth | |
| | d. Hypopituitary and hypothyroid cases | Eruption retarded concomitant with decrease in vascularity of periodontal tissues | |
| | e. Hyperpituitary cases | Supra-eruption concomitant with increased vascularity of periodontal tissues | |

TABLE III

EXPERIMENTAL EVALUATION OF THEORIES OF ERUPTION LISTED IN TABLE I

| | EXPERIMENTAL PROCEDURES IN CONTINUOUSLY ERUPTING RAT INCISOR | FINDINGS | CONCLUSIONS |
|---|---|---|---|
| 1. | a. Cutting off of basal end of tooth and removal of odontogenic epithelium | Root elongation ceases. Eruption continues unaffected until tooth is exfoliated | Eruption of tooth not due to elongation of root |
| | b. Hypophysectomy | Eruption markedly retarded but elongation of root continues and causes "accordion pleating" | |
| 2. | a. Removal of pulp | Dentine growth ceases in pulpal direction. Eruption unaffected | Eruption of tooth not due to growth of dentine or pulpal constriction |
| | b. Hypophysectomy | Eruption markedly retarded but growth of dentine and pulpal constriction continues with dentinification of pulp | |
| 3. | a. Histologic examination of erupting tooth | Periodontal fiber direction indicates that tooth pulls upon periodontal fibers and through them upon alveolar bone during eruption; not vice versa | Organization of periodontal fibers and growth of alveolar bone is secondary to eruption of tooth |
| | b. Extraction of tooth | Alveolar bone resorbs | |
| 4. | No experimental data available | | |
| 5. | Alveolar crest most active site of bone apposition | | Eruption cannot be due to resorption of alveolar crest exposing the tooth into the oral cavity |
| 6. | a. Removal of odontogenic epithelium<br>b. Removal of pulp | Eruption continues | Eruption is not due to proliferation of cells of odontogenic epithelium or pulp |
| 7A. | a. Removal of pulp<br>b. Hypophysectomy | See No. 2 above | Eruption not due to vascularity in pulp |
| 7B. | a. Hypophysectomy | Eruption markedly retarded concomitant with reduced vascularity of periodontal tissues | Eruption of teeth related to vascularity of periodontal tissues |
| | b. Removal of vasoconstrictor nerve | Eruption accelerated concomitant with increased vascularity of periodontal tissues | |

3. Supra-eruption of a tooth will occur, when its antagonist is removed by extraction or bite opening, long after root formation is completed.

4. The distance travelled by some teeth (upper cuspids) during eruption is much greater than the total length of their roots.

5. In many lower forms, particularly the crocodile, the tooth erupts as a whole, quite independent of any increase in the length of the root (Tomes[32]).

In addition, the following circumstantial evidence may be invoked against this theory:

1. During its eruption, the tooth changes its inclination and direction many times. Such changes cannot be explained by elongation of the roots.

2. The total length of the root is attained *after* clinical eruption of the tooth. About four-fifths of the root is completed when eruption of the tooth is clinically completed.

3. Intraoral x-rays and histologic sections show that the root of the tooth does not reach the bottom of the alveolus during eruption. The root does not push against any solid structure, but appears, rather, to be pushed in an occlusal direction.

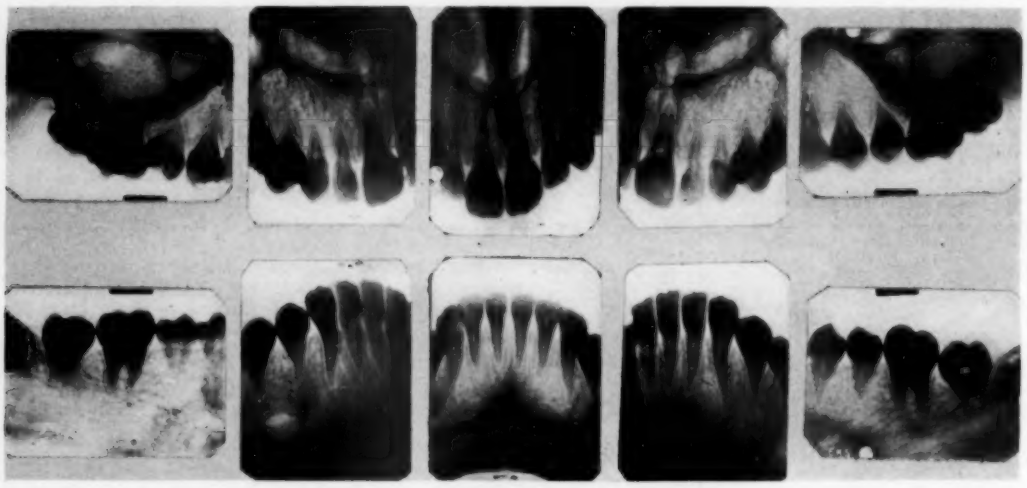


Fig. 3.—Intraoral roentgenograms of a condition characterized by a markedly deficient amount of root formation. This condition may be familial (Ballschmiede, quoted from Landsberger[19]). The eruption time of the teeth was normal although root formation is markedly deficient. (Courtesy of Dr. S. Kloehne.)

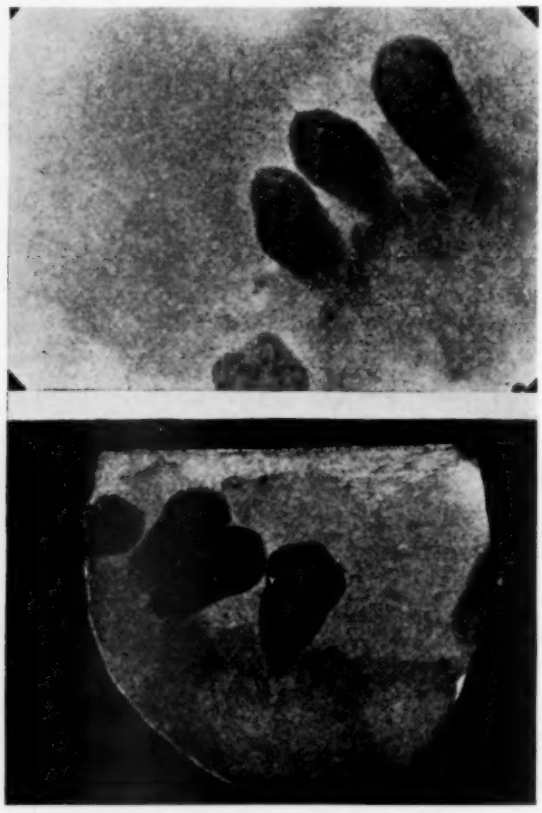


Fig. 4.—Condition similar to that illustrated in Fig. 3, from Landsberger.[19] The crowns with traces of the undeveloped roots may be seen in the roentgenograph.

*Experimental Evaluation:*

*Evidence for Theory.*—None.

*Evidence Against Theory.*—1. Removal of the odontogenic epithelium (Hertwig's epithelial sheath) in the incisor of the rat so as to prevent addition of tooth substance at the basal or apical end does not alter the rate of eruption. There is no "root" elongation, but eruption continues until the tooth is exfoliated (Fig. 5) (Herzberg and Schour[12]).

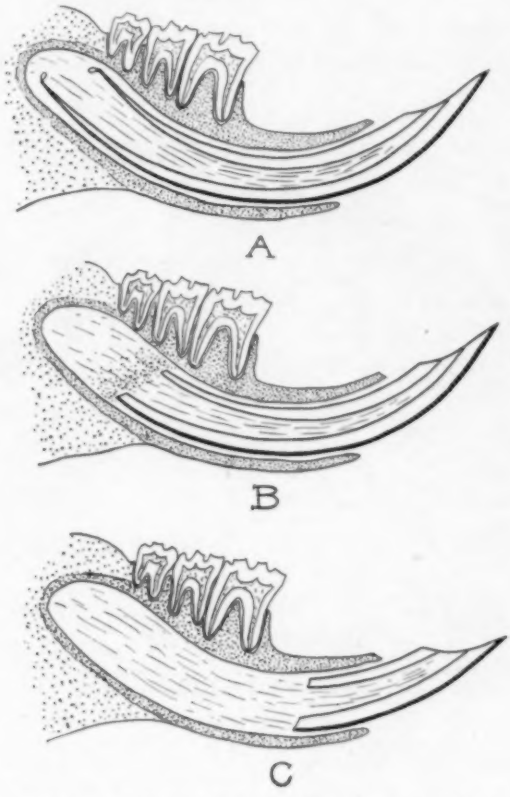



Fig. 5.—Semidiagrammatic tracing of a rat incisor from which the odontogenic epithelium has been removed surgically. (Courtesy of Dr. Herzberg.) *A*, Before operation. *B*, About seven days later. Note the lack of root formation. Eruption rate is normal. *C*, Exfoliation of the tooth about thirty-five days after operation.

2. In hypophysectomy, eruption of the tooth is markedly retarded but elongation of the "root" or embedded portion of the tooth continues, causing the typical plating at the base (Fig. 6) (Schour and van Dyke[30]). The eruption of the tooth and the elongation of its "root" are therefore unrelated and can be dissociated.

3. In the rat molar, in which it has been possible to measure directly the rate of eruption by means of injections of alizarine Red S, it was found that the total amount of eruption was equal to the sum of the amount of apposition of the fundic bone and root elongation (Hoffman and Schour[14]). Thus eruption proceeds at a faster rate than root elongation. This is probably true in the human tooth as well.

4. If root elongation were the primary factor in eruption, the root should push against the bottom of the alveolus and resorption of the periapical bone

would be expected from such pressure. The x-ray and histologic sections show that the root does not reach the bottom of the alveolus and in fact active apposition of bone occurs in that area (Fig. 2). This evidence is, however, only circumstantial.

*Conclusions.*—There is no convincing evidence to support the theory that the elongation of the root is the mechanism whereby the crown of the tooth is pushed into the oral cavity. The clinical and experimental evidences against this theory are many. The theory as it stands, therefore, is inadequate. However, although the elongation of the root cannot be the only factor, the evidence does not exclude it from being one of many factors operating during normal eruption.

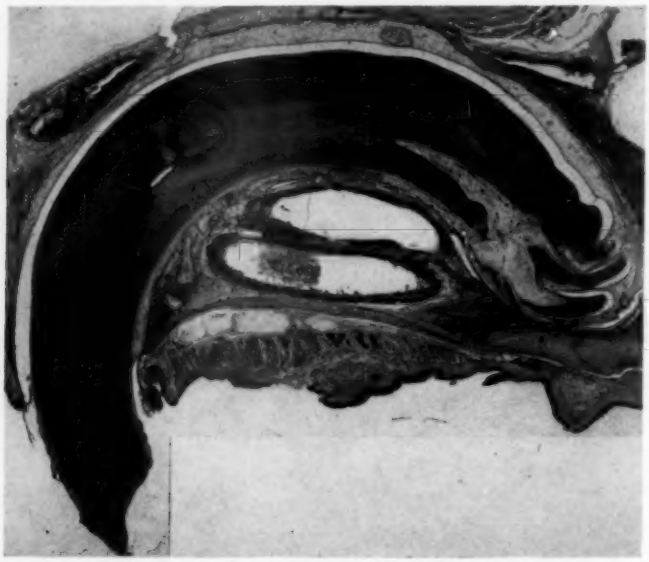


Fig. 6.—Photomicrograph of a sagittal section of an upper incisor from a hypophysectomized rat (×7.2). Eruption has ceased but the continued backward growth of the intra-alveolar dentine has caused a buckling and accordion-like pleating of the basal portion of the tooth.

### 2. PULPAL CONSTRICTION

The theory has been advanced that the growth of the root dentine and the subsequent constriction of the pulp may cause sufficient pressure to move the tooth occlusally (Zuckerkandl, Walkhoff, Eidman, Kallhardt, Eichler, Oehrlein, cited from Weber[33]). Tomes[32] points out that this theory, like the previous one, fails to explain the eruption of rootless or fully formed teeth.

*Clinical Evaluation:*

*Evidence for Theory.*—The pulp is progressively constricted by growth of root dentine as the eruption of the tooth occurs. This evidence is only circumstantial.

*Evidence Against Theory.*—1. Pulpless teeth often erupt at the same rate as their normal neighbors (Fig. 8).

2. A premolar will often "jump" into occlusion after the premature extraction of the deciduous molar without any appreciable growth of dentine or pulpal constriction (Fig. 2).

3. Teeth such as submerged molars and upper cuspids (Fig. 7) often erupt long after dentine formation is completed; the pulps are fully enclosed and the apical foramen is almost obliterated.

4. Supra-eruption will occur when an antagonist is removed by extraction or bite plane long after dentine formation is ended and the apical foramen is quite small, and even in pulpless teeth (Fig. 8).

*Experimental Evaluation:*

*Evidence for Theory.*—None.

*Evidence Against Theory.*—1. Removal of the pulp in the continuously erupting incisor of the rat does not alter the rate of its eruption, although dental formation and pulpal constriction cease (Fig. 9) (Herzberg and Schour[12]).

2. In hypophysectomy, eruption of the rat incisor is markedly retarded in spite of the fact that the pulp becomes increasingly constricted and obliterated by dentine growth (Fig. 6). The eruption of the tooth and the pulpal constriction are therefore unrelated.

*Conclusions.*—The theory that the pressure derived from pulpal constriction incident to dentine growth causes the eruption of the tooth is itself inadequate since it fails to explain many factors in eruption and is supported by no actual evidence. However, the evidence against the theory is not sufficiently conclusive to eliminate entirely pulpal constriction as a possible factor in normal eruption.

Fig. 7.

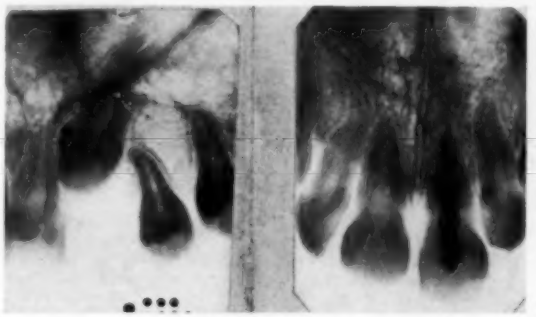


Fig. 8.

Fig. 7.—Roentgenogram of a fully formed cuspid in active eruption. The cuspid came into clinical occlusion about three years after the x-ray was taken. No orthodontic measures were used. (Courtesy of Dr. A. G. Brodie.)

Fig. 8.—Roentgenogram showing the supra-eruption of a pulpless central incisor in a child aged 10 years. Both central incisors were at the same incisal level at the time the pulp was extirpated. Periapical inflammation as a result of mechanical and chemical irritation probably caused the supra-eruption of the treated tooth.

### 3. GROWTH OF PERIODONTAL TISSUES

*A. Pull by Surrounding Connective Tissue.*—Underwood (cited by Tomes[32]) suggests that the connective tissue surrounding the tooth may function in pulling the tooth into the oral cavity. He cites the teeth of continuous succession in fish and reptiles in support of this view. It is not certain whether Underwood intended to apply this theory to the teeth of higher forms. In the latter case, the theory would imply that the periodontal membrane would function to pull the tooth into occlusion. This theory is invalidated by histologic examination of the direction of the periodontal fibers during tooth eruption, which shows that the periodontal fibers are being pulled by the tooth and do not themselves pull upon the tooth (Fig. 10).

*B. Alveolar Bone Growth.*—Hermann[13] and Nessel[22] believed that the growth of the alveolar bone might push or squeeze the tooth out of its alveolus and into the oral cavity. However, x-ray and histologic sections show that the bone does not actually touch the tooth. In addition, this mechanism can operate only upon single conical roots. Multirooted teeth could not, by this mechanism, be erupted. Lastly, as will be discussed in the next paragraph, the growth of the alveolar bone is subservient to and a *resultant* of the movements of the tooth, and not a primary factor.

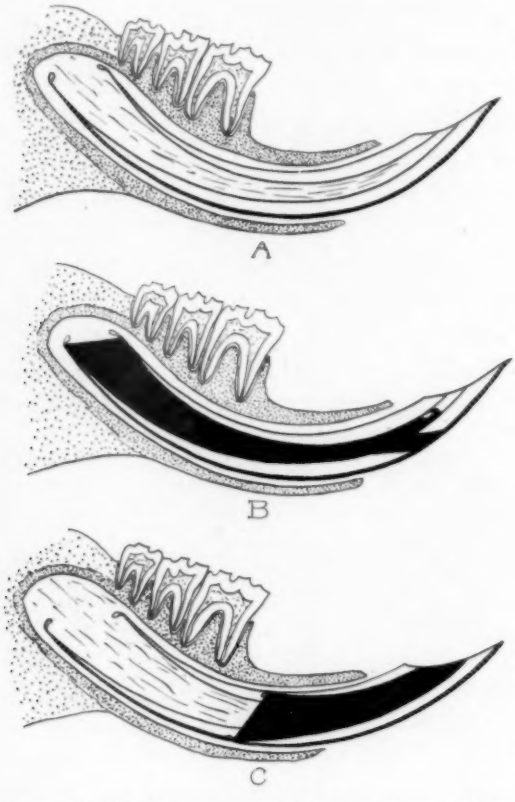



Fig. 9.—Semidiagrammatic tracing of a rat incisor from which the pulp has been removed. Note the lack of dentine formation pulpally in *C*. Eruption rate was normal and equal to the adjacent untreated incisor. (Courtesy of Dr. F. Herzberg.) *A*, Immediately before operation. *B*, Immediately after pulp was removed and the filling with gutta-percha (black). Entrance was made from the labial surface. *C*, About thirty days later. Note the eruption of the pulpless portion of the tooth and the newly formed dentine and pulp from the continuously growing odontogenic base in the distal portion of the tooth.

Brash[2] considers the teeth as purely passive objects whose eruption is incidental to the growth of the alveolar bone. This leads us into a rather paradoxical situation. The growth of the alveolar process is the result of the eruption of the teeth and its existence is entirely dependent upon the presence of the teeth, and yet the teeth are assumed to be passive agents in alveolar bone growth. From the work of Oppenheim and many others, it is apparent that the alveolar bone is formed in response to tooth movement. When a tooth moves it pulls upon the bone through the embedded periodontal fibers. The bone does not either push or pull the tooth. Histologic examination of the periodontal fibers and the bony trabeculae during eruption shows that the migrating tooth is pulling upon the periodontal fibers which are embedded on the one end in

cementum and on the other in the alveolar bone. The orientation of the bony trabeculae shows that eruption provides the stimulus for the growth of the alveolar process (Fig. 10).

Whether the tooth be pulled (or pushed) by the periodontal fibers or by the alveolar bone through the periodontal fibers embedded within it, the effect will be the same. The objections for the one theory are the same as for the other, and therefore will be considered together. These theories are invalid on the following bases:


Fig. 10.—Histologic section of an erupting tooth showing the direction of the periodontal membrane fibers. (From Meyer[21].) During eruption the tooth draws the fibers of the dental periosteum in the direction of movement. Mallory staining. *F*, Fibers of the dental periosteum. *R*, Root. ×40.

1. When a deciduous molar is prematurely extracted, the underlying premolar often jumps out of its crypt and into occlusion. X-ray examination shows that in such cases the eruption is so rapid as to leave the bone far behind (Fig. 2). In fact, the growth of the bone does not catch up with the eruption of the tooth for some time.

2. Histologic examination of human teeth during active eruption shows that the periodontal fibers are so directed as to indicate that the tooth is pulling upon the periodontal fibers embedded within the cementum and the alveolar bone. The orientation of the bony traceculae is such as to indicate also that they are being pulled upon in an occlusal direction and they themselves do not do the pulling (Fig. 11).

The work of Oppenheim[24] and others shows that the growth of the alveolar bone is entirely subservient to and the result of tooth movement rather than the active agent in tooth movement.

3. Vital injections of alizarine red S in the albino rat show that the alveolar bone around the molar teeth forms continually throughout the life of the animal. During the eruption of the teeth, the alveolar crest comes to lie from above the occlusal surfaces of the teeth to below the cementoenamel junction.[14] Therefore the rate of eruption is greater than the rate of alveolar crest formation.

4. Churchill[6] points out that in certain dermoid cysts, the teeth may erupt in the absence of a bony base. Eruption, in such cases at least, cannot be the result of bone growth.


Fig. 11.—Histologic section of the alveolar bone surrounding an erupting tooth. Note the orientation of the trabeculae toward the direction of eruption, indicating the fact that alveolar bone formation occurs as a response to eruption of the teeth, probably by stimulus through the periodontal fibers. (From Orban.) *1*, Pulp; *2*, dentine; *3*, periodontal membrane; *4*, spicules of alveolar bone forming in response to the eruption of the tooth.

*Conclusions.*—Histologically the direction of the periodontal fibers and the bony trabeculae of the alveolar process during eruption show that the tooth is pulling upon the periodontal fibers and, through them, also upon the alveolar bone. Any theory which supposes that the periodontal tissues (connective tissue fibers or bone) are the active agents in pulling or pushing upon the tooth to cause its eruption is unsupported by factual evidence. This does not entirely eliminate, however, the growth of the periodontal tissues as a possible factor in aiding the normal eruption of the tooth.

### 4. PRESSURE FROM MUSCULAR ACTION UPON THE ALVEOLAR PROCESS

It has been suggested by Berten (cited by Demolis[8]) that the action of the musculature of the cheeks and lips upon the alveolar process might serve to

squeeze the crown of the tooth out into the oral cavity like a pumpkin seed from between the fingers. This process continues until the tooth is in occlusion, the process being halted by the antagonism of the teeth. The theory, however, fails to explain why:

1. The teeth (laterals and premolars) erupt which lie lingual to the arch and are therefore under no muscular action of the cheeks and lips.

2. Mouth breathers with notoriously *weak* action of the cheek and lip musculature show a relatively *increased* or supra-eruption of the clinical crowns of the teeth, while people with *strong* musculature and powerful bites show much *less* erupted clinical crowns of the teeth.

3. The eruption of impacted teeth (upper cuspids) proceeds in a horizontal instead of a vertical direction.

4. The teeth erupt in cases of unilateral facial paralysis.

*Conclusion.*—There is no actual evidence to support this theory, and like the others it fails to explain many of the phenomena of eruption. It is highly improbable that the action of the musculature upon the alveolar process might be a factor in causing the eruption of teeth.

### 5. RESORPTION OF ALVEOLAR CREST

Aichel and Weidenreich (cited from Weber[33]) suggest that the resorption of the alveolar crest would serve to expose the crown of the tooth into the oral cavity. This theory is not tenable since histologic examination shows that the alveolar crest is the site of the most rapid and continuous growth of bone (Fig. 12). When resorptions of the alveolar crest occur, periodontoclasia results.

*Conclusion.*—This theory has no basis in fact.

*Hormonal Theory:*

Sir Arthur Keith[16] suggested that the eruption of the teeth may be governed by the hormones secreted by the thyroids and pituitary glands. More recently Kronfeld[18] (citing the work of Schour and van Dyke on the effects of hypophysectomy on the teeth) re-emphasized this hypothesis.

This theory does not attempt to explain the mechanism of the eruption of the teeth, and only points out the fact that the hormones may affect the eruption of the teeth. A relationship is pointed out, but no actual mechanism of action is proposed. The possible mechanism of such action will be discussed in the section on the relation of vascularity to the eruption of the teeth.

*Foreign Body Theory:*

In the same category as the hormonal theory is Gottlieb's foreign body theory, which states that a calcified body such as the tooth tends to be exfoliated by the tissues just as does any foreign body. That a foreign body is extruded is an observable fact, but the mechanism whereby it is caused to move through the tissues is still not clear. Gottlieb[11] merely calls the tooth a foreign body since it is extruded like one. The mechanism whereby it is extruded is not indicated.

### 6. CELLULAR PROLIFERATION

Noyes[23] points out that the tremendous pressure which is evolved from cellular proliferation provides the growing plant with sufficient force to break through stones and other hard obstacles. Eidmann[10] suggests that similarly, the

osmotic pressure and other forces resulting from cellular proliferation in the pulp and surrounding tissues may account for the eruption of the teeth. Histologically, cellular proliferation is confined mainly to Hertwig's epithelial sheath during tooth development. Like the other theories, this theory fails to explain (1) the eruption of teeth long after the proliferation of Hertwig's epithelial sheath is completed and its cells are disintegrated; or (2) the eruption of the rat incisor after Hertwig's epithelial sheath is removed surgically (Herzberg and Schour[12]).

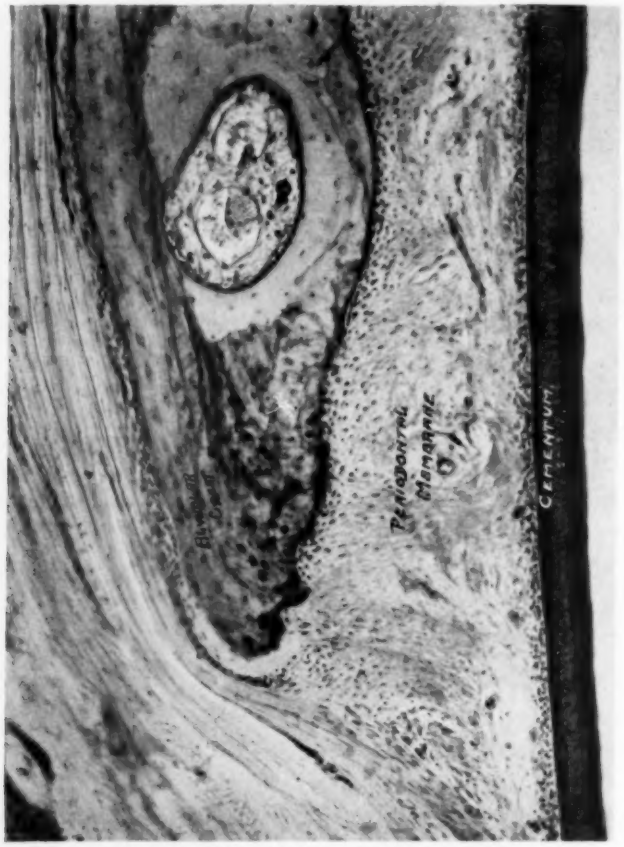



Fig. 12.—Histologic section of the alveolar crest bone surrounding an erupting upper lateral incisor. Note the extremely active apposition of bone in this area. (×92.) (Courtesy of Dr. J. Weinmann.)

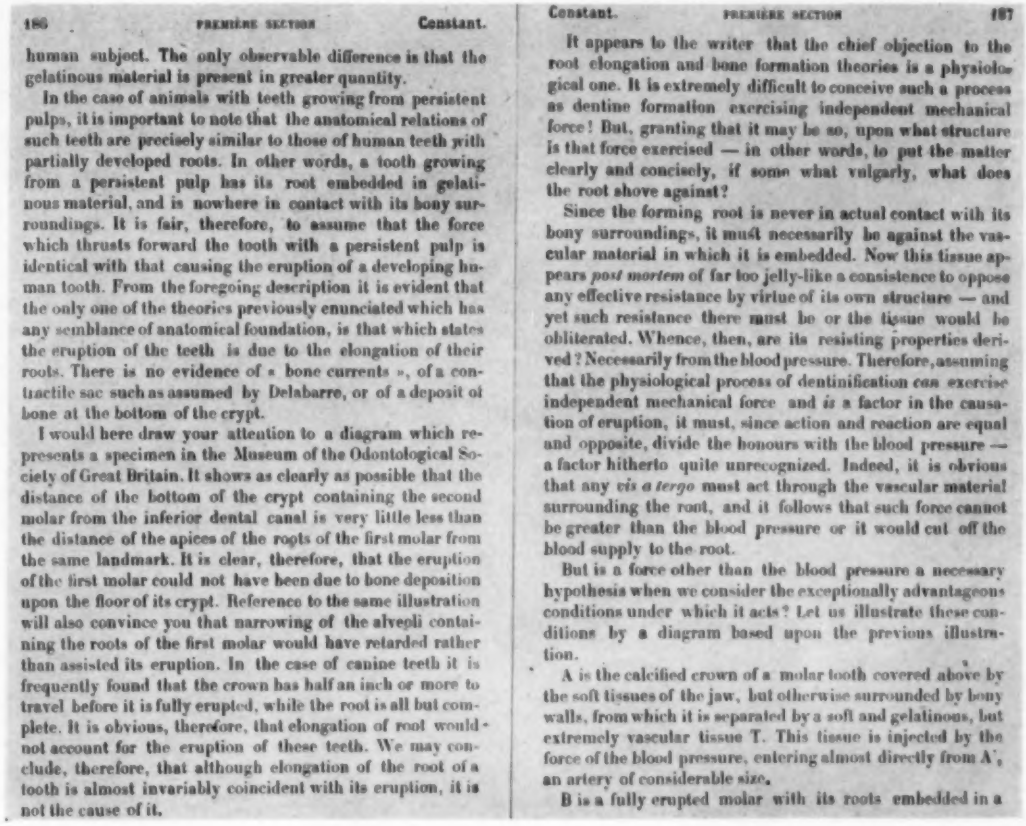
Baume (cited from Weber[33]) believes that the cellular proliferation in the marrow spaces surrounding the tooth causes a tension that in turn causes the eruption of the tooth. The amount of cellular proliferation found in these spaces can hardly account, however, for the relatively tremendous force of eruption.

*Conclusions.*—There are many objections to the theory that cellular proliferation is responsible for tooth eruption. In addition, there is little objective histologic evidence in its support. Cellular proliferation, however, may be one of many factors operating to cause the eruption of teeth.

### 7. VASCULARITY

Constant[7] suggested that the blood pressure may be the impelling force in eruption. He points out the fact that the tissues which lie between the develop-

ing tooth and its bony surroundings possess a very rich vascular supply. Constant's paper has been generally overlooked in the literature, but his concise and clear statement of the problem as well as his logical presentation merits the reproduction of at least part of his original paper (Figs. *A* and *B*).



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human subject. The only observable difference is that the gelatinous material is present in greater quantity.

In the case of animals with teeth growing from persistent pulps, it is important to note that the anatomical relations of such teeth are precisely similar to those of human teeth with partially developed roots. In other words, a tooth growing from a persistent pulp has its root embedded in gelatinous material, and is nowhere in contact with its bony surroundings. It is fair, therefore, to assume that the force which thrusts forward the tooth with a persistent pulp is identical with that causing the eruption of a developing human tooth. From the foregoing description it is evident that the only one of the theories previously enunciated which has any semblance of anatomical foundation, is that which states the eruption of the teeth is due to the elongation of their roots. There is no evidence of « bone currents », of a contractile sac such as assumed by Delabarre, or of a deposit of bone at the bottom of the crypt.

I would here draw your attention to a diagram which represents a specimen in the Museum of the Odontological Society of Great Britain. It shows as clearly as possible that the distance of the bottom of the crypt containing the second molar from the inferior dental canal is very little less than the distance of the apices of the roots of the first molar from the same landmark. It is clear, therefore, that the eruption of the first molar could not have been due to bone deposition upon the floor of its crypt. Reference to the same illustration will also convince you that narrowing of the alveoli containing the roots of the first molar would have retarded rather than assisted its eruption. In the case of canine teeth it is frequently found that the crown has half an inch or more to travel before it is fully erupted, while the root is all but complete. It is obvious, therefore, that elongation of root would not account for the eruption of these teeth. We may conclude, therefore, that although elongation of the root of a tooth is almost invariably coincident with its eruption, it is not the cause of it.

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It appears to the writer that the chief objection to the root elongation and bone formation theories is a physiological one. It is extremely difficult to conceive such a process as dentine formation exercising independent mechanical force! But, granting that it may be so, upon what structure is that force exercised — in other words, to put the matter clearly and concisely, if some what vulgarly, what does the root shove against?

Since the forming root is never in actual contact with its bony surroundings, it must necessarily be against the vascular material in which it is embedded. Now this tissue appears *post mortem* of far too jelly-like a consistence to oppose any effective resistance by virtue of its own structure — and yet such resistance there must be or the tissue would be obliterated. Whence, then, are its resisting properties derived? Necessarily from the blood pressure. Therefore, assuming that the physiological process of dentinification *can* exercise independent mechanical force and *is* a factor in the causation of eruption, it must, since action and reaction are equal and opposite, divide the honours with the blood pressure — a factor hitherto quite unrecognized. Indeed, it is obvious that any *vis a tergo* must act through the vascular material surrounding the root, and it follows that such force cannot be greater than the blood pressure or it would cut off the blood supply to the root.

But is a force other than the blood pressure a necessary hypothesis when we consider the exceptionally advantageous conditions under which it acts? Let us illustrate these conditions by a diagram based upon the previous illustration.

A is the calcified crown of a molar tooth covered above by the soft tissues of the jaw, but otherwise surrounded by bony walls, from which it is separated by a soft and gelatinous, but extremely vascular tissue T. This tissue is injected by the force of the blood pressure, entering almost directly from A', an artery of considerable size.

B is a fully erupted molar with its roots embedded in a

Fig. A.

The extreme vascularity of the basal tissue is perhaps comparable to erectile tissue, and its functional effect has been compared to that of a hydraulic chamber.[18*] Tomes[32] agrees that "the blood pressure, keeping up a state of general tension, may operate to push a solid body in any direction in which there is a diminished resistance, to take up, so to speak, any unoccupied space." In addition, Tomes[32] recognized the fact that a concomitant resorption of structures lying in the path of the erupting tooth will provide the space for the erupting tooth.

The relation between vascularity and the eruption of the teeth has been suggested many times since publication of the article by Constant.[7]

*Clinical Evidence for the Relation of Vascularity to the Eruption of Teeth:*

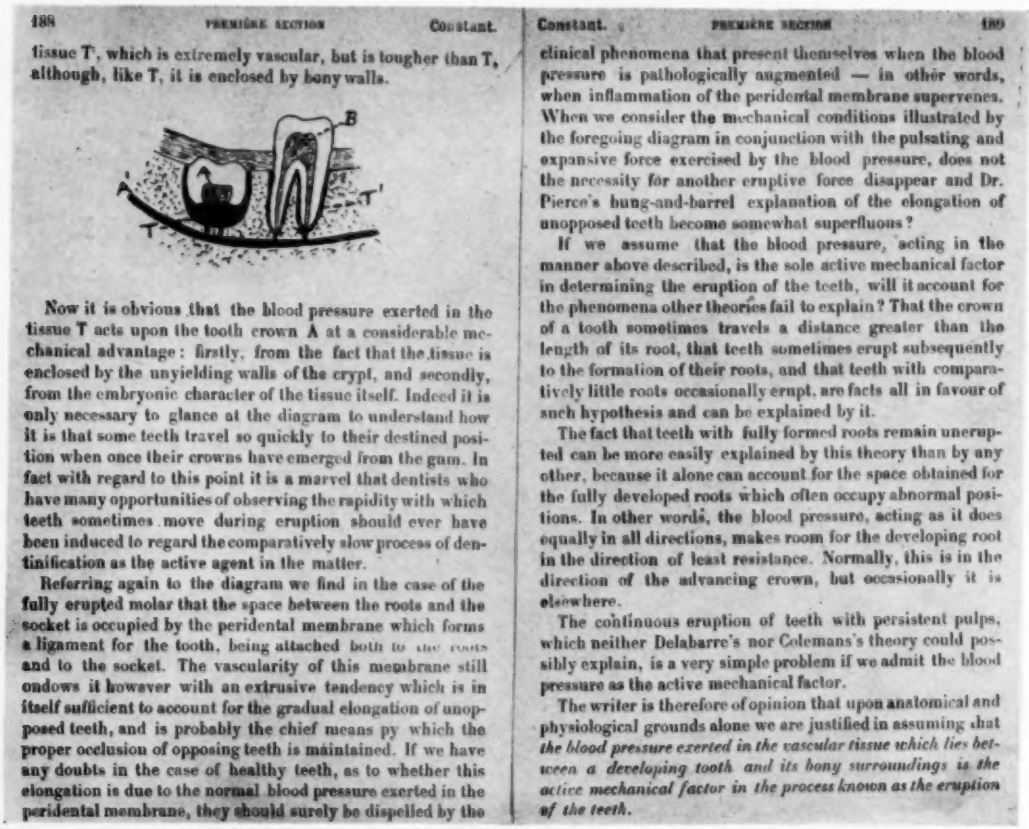
1. Submerged teeth often erupt under the influence of hyperemia induced by mechanical irritations. Thus, a submerged tooth will erupt after an artificial

*Boyle has noted the extremely rich vascularity at the basal end of the continuously erupting incisor of the rat. He likens the effect of this vascularity to a hydraulic chamber which resists occlusal impacts and shows quite clearly the potential force contained within the vascular periapical area.

denture is placed. Examination shows that the tissue is markedly hyperemic. Submerged teeth or teeth delayed in eruption may be accelerated in their eruption by instructing the patient to rub the overlying and surrounding gingival tissues vigorously or by prescribing a diet containing coarsely granular materials to produce the same effect.[4]

2. The hyperemia in periodontitis causes a supra-eruption of the teeth.

3. There is a marked hyperemia or congestion of the periodontal tissues which normally accompanies the eruption of teeth in children (Fischer, Mahé, cited by Demolis[8]). This of course does not prove a cause and effect between the two. The reverse may be the true course of events; that is, eruption of the teeth may cause a congestion of the periodontal tissues.



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tissue T, which is extremely vascular, but is tougher than T, although, like T, it is enclosed by bony walls.



Now it is obvious that the blood pressure exerted in the tissue T acts upon the tooth crown A at a considerable mechanical advantage: firstly, from the fact that the tissue is enclosed by the unyielding walls of the crypt, and secondly, from the embryonic character of the tissue itself. Indeed it is only necessary to glance at the diagram to understand how it is that some teeth travel so quickly to their destined position when once their crowns have emerged from the gum. In fact with regard to this point it is a marvel that dentists who have many opportunities of observing the rapidity with which teeth sometimes move during eruption should ever have been induced to regard the comparatively slow process of dentinification as the active agent in the matter.

Referring again to the diagram we find in the case of the fully erupted molar that the space between the roots and the socket is occupied by the peridental membrane which forms a ligament for the tooth, being attached both to the roots and to the socket. The vascularity of this membrane still endows it however with an extrusive tendency which is in itself sufficient to account for the gradual elongation of unopposed teeth, and is probably the chief means by which the proper occlusion of opposing teeth is maintained. If we have any doubts in the case of healthy teeth, as to whether this elongation is due to the normal blood pressure exerted in the peridental membrane, they should surely be dispelled by the

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clinical phenomena that present themselves when the blood pressure is pathologically augmented — in other words, when inflammation of the peridental membrane supervenes. When we consider the mechanical conditions illustrated by the foregoing diagram in conjunction with the pulsating and expansive force exercised by the blood pressure, does not the necessity for another eruptive force disappear and Dr. Pierce's bung-and-barrel explanation of the elongation of unopposed teeth become somewhat superfluous?

If we assume that the blood pressure, acting in the manner above described, is the sole active mechanical factor in determining the eruption of the teeth, will it account for the phenomena other theories fail to explain? That the crown of a tooth sometimes travels a distance greater than the length of its root, that teeth sometimes erupt subsequently to the formation of their roots, and that teeth with comparatively little roots occasionally erupt, are facts all in favour of such hypothesis and can be explained by it.

The fact that teeth with fully formed roots remain unerupted can be more easily explained by this theory than by any other, because it alone can account for the space obtained for the fully developed roots which often occupy abnormal positions. In other words, the blood pressure, acting as it does equally in all directions, makes room for the developing root in the direction of least resistance. Normally, this is in the direction of the advancing crown, but occasionally it is elsewhere.

The continuous eruption of teeth with persistent pulps, which neither Delabarre's nor Colemans's theory could possibly explain, is a very simple problem if we admit the blood pressure as the active mechanical factor.

The writer is therefore of opinion that upon anatomical and physiological grounds alone we are justified in assuming that *the blood pressure exerted in the vascular tissue which lies between a developing tooth and its bony surroundings is the active mechanical factor in the process known as the eruption of the teeth.*

Fig. B.

4. Following an intra-alveolar fracture, the incisal segment continues to erupt while the apical segment becomes ankylosed. The site of fracture becomes markedly hyperemic. This hyperemia may be the prime factor in causing the migration of the incisal segment (Figs. 13 and 14). There appears to be no clinical evidence against this theory.

5. Circumstantial evidence may be invoked to support the relationship between vascularity and the eruption of teeth. The continuously erupting teeth of the herbivore show a richly vascular periapical area about the widely open apical foramina, whereas the teeth of the carnivore, which erupt only to a

limited degree even when not in occlusion, show a relatively poorly vascular periapical area about the closed apical foramina.

*Experimental Evidence for the Relation of Vascularity to the Eruption of Teeth:*

1. Leist,[20] Breitner and Leist,[5] and King,[17] by sectioning or destroying the sympathetic innervation to one side of the jaw in pups, kittens, and guinea pigs,

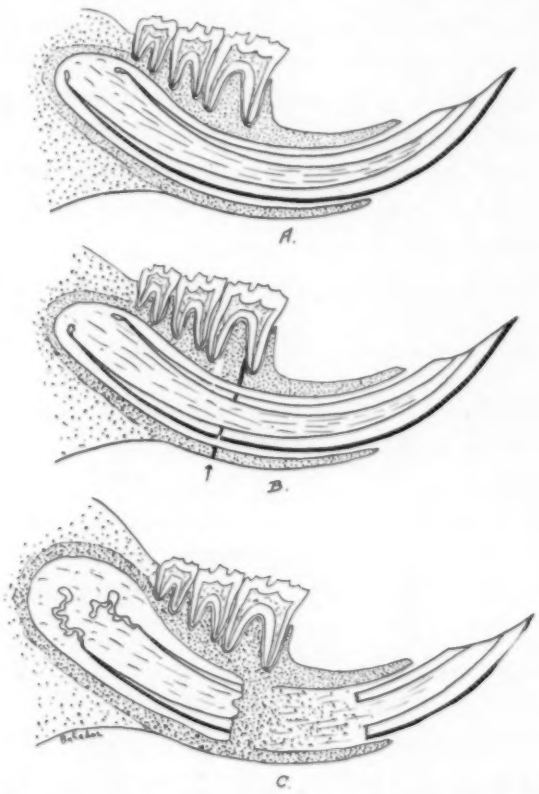


Fig. 13.—Semidiagrammatic tracing of a rat incisor which has been subjected to experimental intra-alveolar fracture. *A*, Before operation. *B*, At time of fracture. Site of fracture indicated by arrow. *C*, About thirty days later. Bone has filled in part of the space between the two segments of the tooth. The basal portion is, as a result, ankylosed. Continued dentine formation at the odontogenic base has resulted in a folding similar to that seen in hypophysectomy. The anterior segment erupted at a fairly normal rate, probably impelled by the inflamed area at its base.

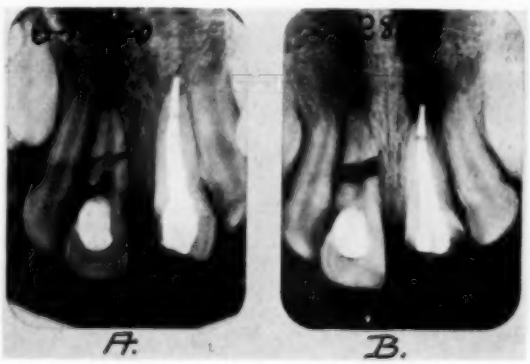


Fig. 14.—Roentgenograms of a fractured upper right central incisor in a 10-year-old child. *A*, X-ray taken about two months after the accident. *B*, X-ray taken nine months later. Note the supra-eruption in *B*, probably caused by an inflammatory reaction at the site of fracture between the coronal and apical segments. (Courtesy of Dr. Elsie Gerlach.)

produced a permanent vasodilatation and marked increase in vascularity (King) on the operated side. They observed also an increased rate of eruption of the teeth on that side. Their work is significant because it offers *experimental* proof in the support of the vascular theory on the mechanism of eruption.

2. Occasionally children with completely submerged teeth or entire dentures are referred to the clinic. In such cases the teeth were exposed by surgical removal of the overlying bone and gingival tissue on one side and the patient was instructed to rub the other side vigorously and to eat coarsely granular foods in order to cause hyperemia. Frequently the teeth on the unoperated side so treated erupt as rapidly as those on the operated side.[31a]

3. In hypophysectomy, the tremendous decrease in the rate of eruption is correlated with a marked decrease in the vascularity of the periodontal tissues.

*Conclusion.*—There is considerable evidence that links the vascularity of the periodontal tissues to the eruption of the teeth. Evidence to the contrary is not apparent.

### LOCALIZATION OF THE ERUPTIVE FORCE

It is as important to localize the area of action of the force responsible for the eruption of the tooth as it is to discover the derivation of such force. The two of course are related.

It is conceivable that the site of the eruptive force might lie either in the highly vascular pulp or in the periapical tissue or in both. That this force is not derived primarily from the highly vascular pulp is shown by the following facts:

1. Eruption continues normally in pulpless teeth and teeth with minute apical foramina and fibrotic pulps.

2. The normal rate of eruption persists in the rat incisor after a complete pulpectomy and filling in with gutta-percha (Fig. 6) (Herzberg and Schour[12]).

3. The rate of eruption is markedly retarded in hypophysectomy in the rat and in hypopituitarism or hypothyroidism in man in spite of wide-open pulp canals.

4. Supra-eruption does not occur in cases of increased pulpal vascularity (pulpitis) while it does in a periodontitis.

*Conclusion.*—Whatever be the source of the eruptive force, it is probably derived from the periapical tissue rather than from the pulp. If the eruptive force is related to vascularity, it must therefore be derived primarily from the richly vascular periapical tissue rather than from the pulp.

## DISCUSSION

### DISSECTION BETWEEN GROWTH AND ERUPTION OF THE TOOTH

A review of the literature shows that the terms "growth" and "eruption" are frequently used interchangeably. The result has been a confusion of the two processes.

In evaluating the various theories on eruption it becomes apparent that the two processes are distinctly different. The growth of the tooth involves an

TABLE IV

SUMMARY OF EVIDENCE FOR AND AGAINST THEORIES OF ERUPTION

| | THEORIES | EVIDENCE | CONCLUSIONS |
|---|---|---|---|
| 1. | Growth of root | a. Eruption occurs in rootless teeth<br>b. Eruption occurs after root formation is completed<br>c. Cuspid erupts a distance more than total length of root<br>d. Eruption continues when odontogenic epithelium is removed and root elongation ceases<br>e. In hypophysectomy eruption ceases but root elongation continues (with foldings) | Theory not valid |
| 2. | Growth of dentine and pulpal constriction | a. Eruption continues in pulpless teeth<br>b. In hypophysectomy, eruption ceases but growth of dentine and pulpal constriction continue | Theory not valid |
| 3. | A. Growth of and pull by periodontal tissue | Histologic examination of erupting tooth shows that erupting tooth is pulling upon periodontal fibers and through them on the alveolar bone; not vice versa | Theory not valid |
| | B. Growth of alveolar bone | a. Bicuspid erupts rapidly with very little growth of alveolar bone when deciduous molar prematurely extracted<br>b. Alveolar bone does not grow but resorbs in absence of tooth<br>c. Teeth in dermoid cysts erupt in absence of bony base | Theory not valid |
| 4. | Pressure from muscular action upon alveolar process | a. Teeth lingual to arch and outside action of cheek musculature erupt at normal rate<br>b. In cases of unilateral facial paralysis, teeth on affected side erupt at normal rate | Theory not valid |
| 5. | Resorption of alveolar bone exposes tooth | Alveolar process *increases* in size during eruption of teeth | Theory not valid |
| 6. | Pressure from cellular proliferation | a. Eruption continues after removal of proliferating odontogenic epithelium<br>b. Number of mitotic figures in periapical tissues not commensurate with relatively tremendous force of eruption | Theory inadequate |
| 7. | A. Pressure from pulp due to:<br>1. Cellular proliferation<br>2. Vascularity, or<br>3. Both | Eruption continues in pulpless teeth. | Theory not valid |
| | B. Pressure due to vascularity of periapical tissues | a. Submerged teeth erupt under the influence of hyperemia induced by mechanical irritation (dentures or finger rubbing)<br>b. Hyperemia in periodontitis causes supra-eruption of tooth<br>c. In hypopituitarism and hypothyroidism eruption is markedly retarded concomitant with reduced vascularity of periodontal tissues. In hyperpituitarism, eruption is accelerated and vascularity of periodontal tissues increased<br>d. Removal of vasoconstrictor nerve causes accelerated eruption concomitant with increased vascularity of periodontal tissues | Theory valid.<br>Eruption of teeth apparently related to vascularity of periapical tissues |

increase in the size of the tooth substances (enamel, dentine, cementum) as a result of cellular activity. Eruption of the tooth involves only the migration of the tooth from one position to another. This is not a growth phenomenon, but the movement of an object—the tooth—through tissues. Each process is separate and distinct from the other and can easily be dissociated clinically and experimentally.

*Clinical Dissociation.*—1. Eruption of teeth can occur long after the growth of enamel and dentine is completed.

2. Premature extraction of a deciduous molar often causes a marked acceleration in the rate of eruption of the premolar tooth without any concomitant acceleration of the growth of the dentine.

*Experimental Dissociation.*—1. In hypophysectomy and in the placing of high crowns, eruption is retarded systemically or mechanically, but the growth of the dentine is not similarly affected.

2. Cutting off of the clinical crown of the incisor of the rat results in a marked acceleration in the rate or eruption but no effect upon the rate of dentine growth (Hoffman[15]).

3. Removal of the odontogenic epithelium or the pulp results in a cessation of growth of dentine pulpally and apically, but eruption continues.

*Summary.*—The eruption of the tooth is not a growth process and can be readily dissociated from the growth of the dental tissues. While the eruption of the tooth is usually synchronized with its growth, just as calcification is usually synchronized with growth, it is of itself quite an independent developmental process. In using the term "eruption" we should confine ourselves strictly to the *migration* of the tooth.

## RELATION OF SYSTEMIC FACTORS TO THE RATE OF ERUPTION

The influence of *local* or *mechanical* factors upon the rate of eruption has already been mentioned. Many similar examples of the effects of local factors upon eruption may be observed in the teeth of children.

It is interesting in addition to observe clinically the relation of various *systemic* factors to the rate of eruption of the teeth and to speculate upon the possible mechanism whereby such a relationship can be effected.

*Influence of Sex.*—Girls erupt their teeth clinically at a more rapid rate and at an earlier chronologic age than do boys. This of course is correlative with the earlier and more rapid general body growth and development of the female during the childhood period. For example, the first permanent molar usually appears in the oral cavity in girls by 5½ years and at least 6 months later in boys. The central incisors appear in girls by 6½ years and in boys 6 to 9 months later. This is quite evident in fraternal twins where the genetic and environmental background is similar and only the sex is different.

The correlation with general body growth does not of course explain the mechanism whereby the teeth are caused to erupt earlier in the female. When one has observed the profound effects of hypothyroidism, hypopituitarism and the debilitating diseases upon the eruption of the teeth and the concomitant

effects upon the basal metabolic rate and vascularity of the tissues one is tempted to relate the eruption of the teeth not only to general health but more specifically to cellular metabolism and tissue vascularity. It would be interesting and instructive to determine the relation of sex to basal metabolism and to tissue vascularity.

*Influence of Health and Disease.*—When brothers or sisters or even identical twins (with the same genetic and environmental background) are observed clinically, a difference in the rate of eruption is often evident.

It is observed sometimes that the robust, vivacious child erupts his teeth at an earlier age than does his more stolid and less active brother. When identical twins are observed, the more active and vivacious of the pair often shows an earlier and more rapid rate of eruption.

The effects of disease on eruption are even more marked than constitutional vitality. We have had the opportunity to observe both the healthy and the sick children of a given family through the cooperation of the Departments of Pediatrics and Children's Dentistry at the University of Illinois. The effect of chronic heart disease is quite marked, the rate of eruption of the teeth being retarded as compared with the dentition of a normal and younger brother. The child who has experienced a long series of debilitating diseases also shows a retarded eruption when compared with his healthy normal brother.

On the other hand, pediatricians have observed that children who suffer from exanthematous diseases tend to erupt their teeth at an earlier age and at a more rapid rate than do their normal healthy brothers.

Such observations do not prove—but do suggest—a relation of the teeth to the body metabolism and tissue vascularity.

It is not justified to draw conclusions from empiric observations which are not yet subjected to experimental discipline. However, we are permitted such speculation on the basis that while the relation of these factors to the eruption of the teeth may be ultimately disproved, at the present time it affords a theoretical mechanism which we might use in our analytical thinking. By so doing we can test the validity of this theory.

## SUMMARY AND CONCLUSIONS

1. A review of the clinical and experimental evidence for and against the many current theories of eruption reveals considerable circumstantial and a small amount of factual evidence against all current theories except those that relate eruption to the vascularity of the periapical tissues (Table IV). There is, on the other hand, considerable circumstantial evidence but no factual proof in favor of the theory that the vascularity of the periodontal tissues is an important factor in the causation of eruption. Some of the evidence in support of this theory is:

A. When the tissues surrounding submerged teeth are caused to increase their vascularity, those teeth tend to erupt. For example, submerged teeth under a denture will erupt when the tissue is irritated enough to cause a hyperemia. Submerged teeth, single or multiple, when rubbed with the finger so as

TABLE V

FACTORS WHICH MAY INFLUENCE THE RATE OF ERUPTION OF TEETH

| | LOCAL FACTORS | SYSTEMIC FACTORS | | |
|---|---|---|---|---|
| | | CONSTITUTIONAL FACTORS | ENDOCRINE FACTORS | NUTRITIONAL FACTORS |
| ACCELERATION (++) | Premature extraction of deciduous predecessor and loss of antagonist (++)<br>Local hyperemia (++)<br>Periodontitis<br>Coarse foods | Diseases causing an increased blood pressure and B. M. R. (++)<br>Prolonged fevers<br>Healthy and robust (+) | Hyperpituitarism (++++)<br>Tumor of adrenal cortex (++) | |
| NORMAL (+−) | | Female<br>Male | | |
| RETARDATION (−−) | Mechanical obstructions (−−)<br>Impactions<br>Ankylosis<br>Bone or fibrous scar over crown<br>Badly twisted root | Puny and stolid (−)<br>Diseases causing a lowered blood pressure and B. M. R. (−−)<br>Chronic cardiac insufficiency<br>Hibernation (−−−−) | Hyperthyroidism (−−−)<br>Hypopituitarism (−−−−) | Fluorosis (−)<br>Vitamin A deficiency (−)<br>Severe multiple avitaminosis (−−)<br>Mg. deficiency (−−) |

to cause a hyperemia, tend to erupt as fast as when similar teeth are freed by operative procedure. Inflammation of the periapical tissue causes a "rising up" or supra-eruption of the tooth.

B. Experimental elimination of the vasoconstrictor action causes an increase in vascularity and concomitant acceleration in eruption.

C. The effects of hormones and other systemic factors upon the eruption of the teeth (the pituitary and thyroid) may be explained through the concomitant effects of these hormones upon the vascularity of the tissues surrounding the teeth.

We therefore are obliged to conclude, at least tentatively, that the eruptive force, or the force responsible for the tooth movement during eruption, may be related to the vascularity of the tissue which surrounds the tooth.

2. Whatever the ultimate source of the eruptive force, clinical and experimental evidence shows that it is probably located in the periapical region of the tooth. The movement of the tooth during eruption apparently results from a force operating at and about its base which tends to drive the tooth in an occlusal direction. This direction can, however, be disturbed by mechanical obstructions.

3. A distinction should be made between the growth and the eruption of the tooth. The two are distinctly separate developmental processes and can be dissociated clinically and experimentally.

4. While at the present time the evidence presented appears to favor the vascularity of the tissues as the source of the eruptive force, such evidence is not at all conclusive—only provocative. It will be necessary in the future to

construct experiments specifically designed to increase or decrease the vascularity of the tissues surrounding the tooth and to watch the effects upon the eruption. Such experiments might include:

A. Reducing the blood supply to one side of the jaw in a young animal.

B. Raising the blood pressure (over a long period of time) *systemically* or *locally*, or both.

5. The evidence presented in this report *does not eliminate other factors or theories* as supplementary agents or sources of the eruptive force. As a matter of fact, the eruptive force probably is not derived simply from one source but may be the resultant of many factors. No conclusive proof could be obtained in favor of any one theory of eruption. The solution of the problem as to the mechanism of eruption therefore awaits further experimental and clinical investigation.

REFERENCES


1. Boyle, P. E.: Tooth Suspension; A Comparative Study of the Paradental Tissues of Man and of the Guinea Pig, J. Dent. Res. **17**: 37, 1938.
2. Brash, J. C.: The Growth of the Alveolar Bone and Its Relation to the Movements of the Teeth, Including Eruption, INT. J. ORTHODONTIA, ORAL SURG. & RADIOGRAPH. **14**: 196, 283, 398, 487, 1928.
3. Brodie, A. G.: Present Status of Our Knowledge Concerning Movement of the Tooth Germ Through the Jaw, J. A. D. A. **24**: 1830, 1934.
4. Brodie, A. G.: Personal communication.
5. Breitner, C., and Leist, M.: Über den Einfluss des Vegetativen Nervensystems auf die Zähne, Ztschr. f. Stomat. **25**: 772, 1927.
6. Churchill, H. R.: In Meyer's Normal Histology and Histogenesis of the Human Teeth and Associated Parts (p. 241), translated and edited by H. R. Churchill, Philadelphia, 1935, J. B. Lippincott Co.
7. Constant, T. E.: The Eruption of the Teeth, Third Congrès Dentaire Internat., Paris **2**: 180, 1900.
8. Demolis, P.: Étude histologique du modelage de l'alveole et l'éruption dentaire, Schweiz. Mschr. f. Zahnheilk. **36**: 85-148, 161-190, 1926.
9. Eichler, M.: Dentition, In Scheff's Handb. Zahnhlk. **1**: 530, 1922.
10. Eidmann, H.: Die Entwicklungsgeschichte der Zähne des Menschen, Berlin, 1923, Meusser.
11. Gottlieb, B.: Rachitis and Enamel Hypoplasia, Dental Cosmos **62**: 1209, 1920.
12. Herzberg, F., and Schour, I.: Effects of the Removal of Pulp and Hertwig's Sheath on the Eruption of Incisors in the Albino Rat, J. Dent. Res., 1941. In press.
13. Hermann, E.: Einiges über die Vorgänge bei der Zweiten Zahnung, Inaug. Dissertation, Halle, 1869 (cited by Demolis).
14. Hoffman, M. M., and Schour, I.: Quantitative Studies in the Development of the Rat Molar. II. Alveolar Bone, Cementum and Eruption (from Birth to 500 Days), AM. J. ORTHODONTICS AND ORAL SURG. **26**: 854, 1940.
15. Hoffman, M. M.: Experimental Alterations in the Rate of Eruption of the Rat Incisor, Master's Thesis, Univ. of Illinois Graduate School, 1939.
16. Keith, A.: An Inquiry Into the Nature of the Skeletal Changes in Acromegaly, Lancet **1**: 993, 1911.
17. King, J. D.: Dietary Deficiency, Nerve Lesions and the Dental Tissues, J. Physiol. **88**: 62, 1936.
18. Kronfeld, R.: Histopathology of the Teeth and Their Surrounding Structures, Philadelphia, 1939, Lea & Febiger.
19. Landsberger, R.: Histologic Research Concerning the Growth of the Alveolar Process in Its Relation to the Development of the Tooth Germ, Dental Cosmos **66**: 1334, 1924.
20. Leist M.: Über den Einfluss des vegetativen Nervensystems auf die Zähne, Ztschr. f. Stomat. **25**: 765, 1927.
21. Meyer's Normal Histology and Histogenesis of the Human Teeth and Associated Parts, translated and edited by H. R. Churchill, Philadelphia, London, and Montreal, 1935, J. B. Lippincott Co.
22. Nessel, F.: Compendium der Zahnheilkunde, Wien, 1856. (Cited from Demolis.)
23. Noyes, F. B., Schour, I., and Noyes, H. J.: Dental Histology and Embryology, Philadelphia, 1938, Lea & Febiger.
24. Oppenheim, A.: Tissue Changes, Particularly of the Bone, Incident to Tooth Movement, Am. Orthodont. **3**: Nos. 2 & 3, 1912.

25. Orban, B.: Dental Histology and Embryology, Philadelphia, 1929, P. Blakiston's Son & Co.
26. Reichborn-Kjennerud, I.: Über die Mechanik des Durchbruches der Bleibenden Zähne beim Menschen, Berlin, 1934, Meusser.
27. Sarnat, B. G., and Hook, W. E.: Effect of Hibernation on Rate of Eruption and Dentin Apposition in the Ground Squirrel Incisor, Proc. Soc. Exper. Biol. & Med. **46**: 685, 1941.
28. Schour, I.: The Hypophysis and the Teeth. I. Changes in the Rat Incisor Following Hypophysectomy, Angle Orthodontist **4**: 3, 1934.
29. Schour, I., Brodie, A. G., and King, E. Q.: The Hypophysis and the Teeth. IV. Dental Changes in a Hypopituitary Condition. A Case Report, Angle Orthodontist **4**: 285, 1934.
30. Schour, I., and van Dyke, H. B.: Changes in the Teeth Following Hypophysectomy. I. Changes in the Incisor of the White Rat, Am. J. Anat. **50**: 397, 1932.
31. Schour, I., and Massler, M.: 1940. The Effect of the Endocrines on the Teeth, Jaws and Facial Skeleton, Proc. Dent. Cent., Baltimore, pp. 145-156, March, 1940.
31a. Schultz, L.: Personal communication.
32. Tomes, Sir Charles S.: A Manual of Dental Anatomy (8th ed. by Timms and Henry), New York, 1923, The Macmillan Co.
33. Weber, R.: Dentition, in Kantorowicz Handwörterbuch der Gesanten Zahnheilkunde, 1929, Barth, Leipzig, and Meusser, Berlin.
34. Wetzel, G.: Die Regulationen der Nagetierschneidezähne, Arch. f. Entwklngsmech. *d* Organ. **112**: 455, 1927.

# JOHNSON TWIN ARCH TECHNIQUE AND SPECIAL ARCH PULLING VISE

LOWRIE J. PORTER, D.D.S., NEW YORK, N. Y.

THIS clinic was given to show various gadgets for the making of Johnson twin arches including a new type of vise for pulling the twin wires accurately and quickly through the tubes and, at the same time, controlling the position of the intermaxillary hooks. The latter has caused considerable difficulty in the past as the hooks seemed to vary in position a great deal, necessitating a twisting of the buccal tubes to correct the hook positions.

Fig. 1, *A*, shows a jig for cutting buccal tubes. This jig should be fastened in a vise. Johnson tubing is run into a longer tubing (.040 I.D.) as shown. The large tubing on the left end is cut 1⅛ inches long, acting as a gauge for the cutting of the Johnson tubes. At the left end of this tubing is a flat spring which acts as a stop while the Johnson tubing is being cut as shown by knife-edge stone. The Johnson tubing continues to be fed in after each tube cutting which automatically pushes out the tube which has been cut. (Spring releases sufficiently to allow this.) Many tubes can be cut accurately and in a very short time.

The next step in assembly is the cutting of twin wires as shown in Fig. 1, *B*. These wires are cut 3¼ inches long on a wire cutting jig, shown in Fig. 2. The spool shown on the post is a silk ligature spool trimmed down to make the circumference 6½ inches. Two slots have been cut, as shown on opposite sides of the spool, in order that scissors may be inserted at this point to cut the wire. The knob serves as a handle for winding wire from the large spool. When sufficient wire has been wound, two rubber bands are placed crosswise as shown to keep the wires from being displaced when the wire is cut in the slots by a pair of scissors. This will quickly give many wires cut accurately at 3¼ inches in length. The illustration shows how the wires are held in place after they have been cut. This device is also used for cutting ligature wires to be used at the operating chair.

Step (3) consists of threading the tubes with the twin wires. We have found two twin wires to work better for us than the looped wire which is generally used. It saves bending it back on itself, and we also believe it pulls through the tubes more accurately. A tube threading jig, as shown in Fig. 1, *C*, can be made by using two open tubes held in place by soldering to a frame. The Johnson tubing is placed in the frame and held in place by spring pressure. This brings the ends of two pieces of tubing in contact with each other so that both tubes can be threaded at one time.

If intermaxillary hooks are used, they may be soldered readily by the use of a jig as shown in Fig. 1, *D*. The Johnson tube is placed in the open tubes

Clinic given before the American Association of Orthodontists, New York City, May 6, 1941.

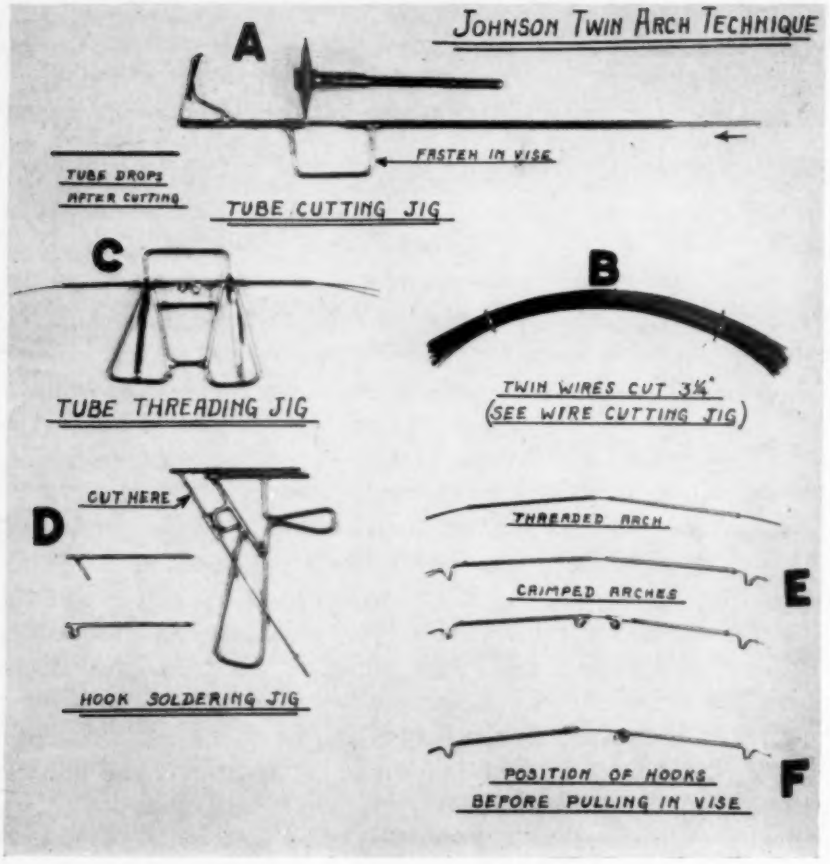
JOHNSON TWIN ARCH TECHNIQUE
A
FASTEN IN VISE
TUBE DROPS AFTER CUTTING
TUBE CUTTING JIG
C
TUBE THREADING JIG
B
TWIN WIRES CUT 3¼"
(SEE WIRE CUTTING JIG)
D
CUT HERE
HOOK SOLDERING JIG
THREADED ARCH
CRIMPED ARCHES
E
POSITION OF HOOKS
BEFORE PULLING IN VISE
F

Fig. 1.

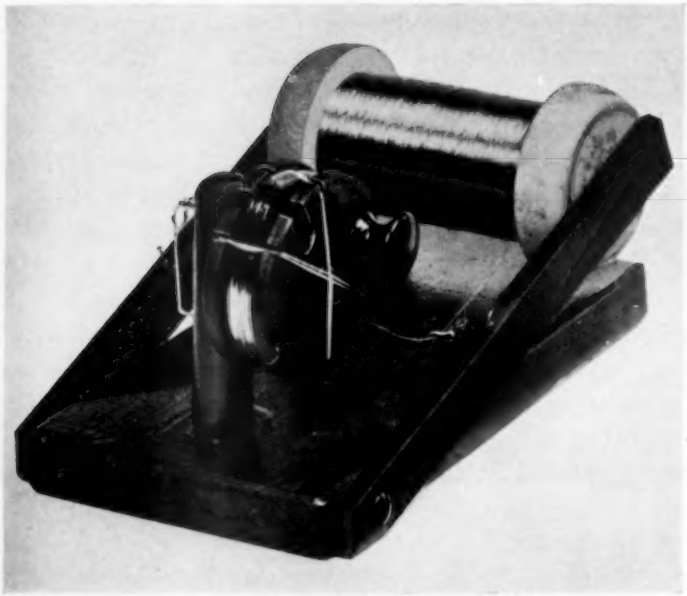

Fig. 2.

shown at the tops of the jig. An 0.023 wire is run through a snug-fitting tube of that size placed on the side of the jig in such a position that the 0.023 wire will contact the tube about $\frac{1}{32}$ of an inch from the tube end. The wire is then soldered and cut off as indicated and then bent into a hook shape. A jig is very essential for this as freehand soldering in stainless steel seems to have resulted in much breakage of hooks, probably because the wire has been moved slightly during the time the solder is chilling which has caused a crystallization of the solder and eventual breakage.

The twin wires are now crimped as shown in Fig. 1, *E*. It is essential that both of these crimps should be in the same direction that is, parallel to each other.

The arch is now placed in the vise designed for this purpose. We find that this vise works much more efficiently than other vises in common use.

This vise (Fig. 3) is designed with a right and left hand thread so that the end blocks pull away from each other and away from the stationary center block to any desired distance. The center section with the thumbscrew grips the twin wires in the center holding them firmly while the tubes are pulled along the twin wires as shown.

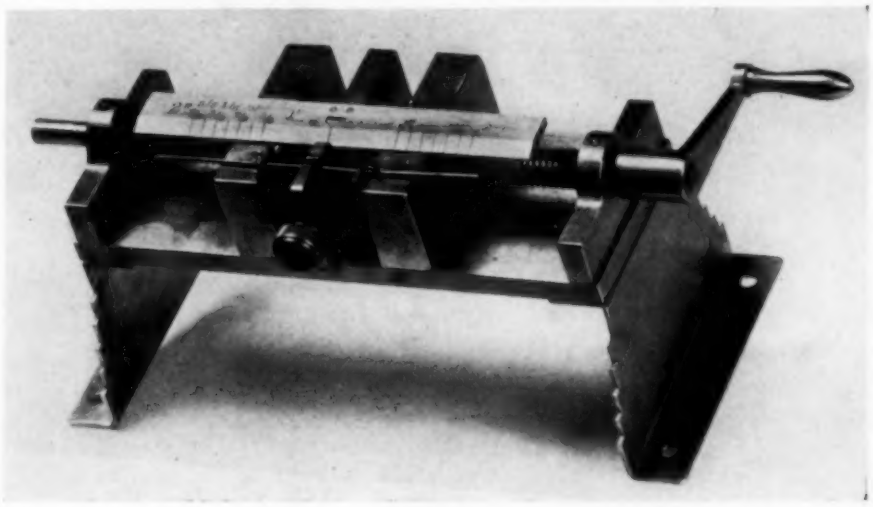

Fig. 3.

By carefully threading the wires through the tubes and crimping them together, there should not be the difficulty of having the anterior twin sectional wires crossed after the arch is pulled. Any size arch may be pulled as indicated on the graduated scale.

It is our custom to have a stock of seven varied sizes of twin arches numbering from one to seven, Fig. 4, *A, B, C*.

The number one arch has a twin wire anterior section measuring $1\frac{3}{8}$ inches. Each successive arch increases a quarter of an inch in the anterior section, making arch number two $1\frac{5}{8}$ inches, the next, $1\frac{7}{8}$ inches etc., until number seven arch is $2\frac{7}{8}$ inches. It will be found that numbers six and seven are very rarely used and could easily be eliminated so far as stocking is concerned.

By the use of this vise, Fig. 3, the twin wires are pulled into both tubes exactly the same distance. Since the twin wires are 3¼ inches long, a twin section pulled 1¼ inches would leave one inch of wire in each tube. Making the size of the twin section longer results in leaving that much less wire in the tubes. Cutting the tubes off will not cause the twin wires to slip out of the tubes unless the tube is cut off so much that the crimped section of the

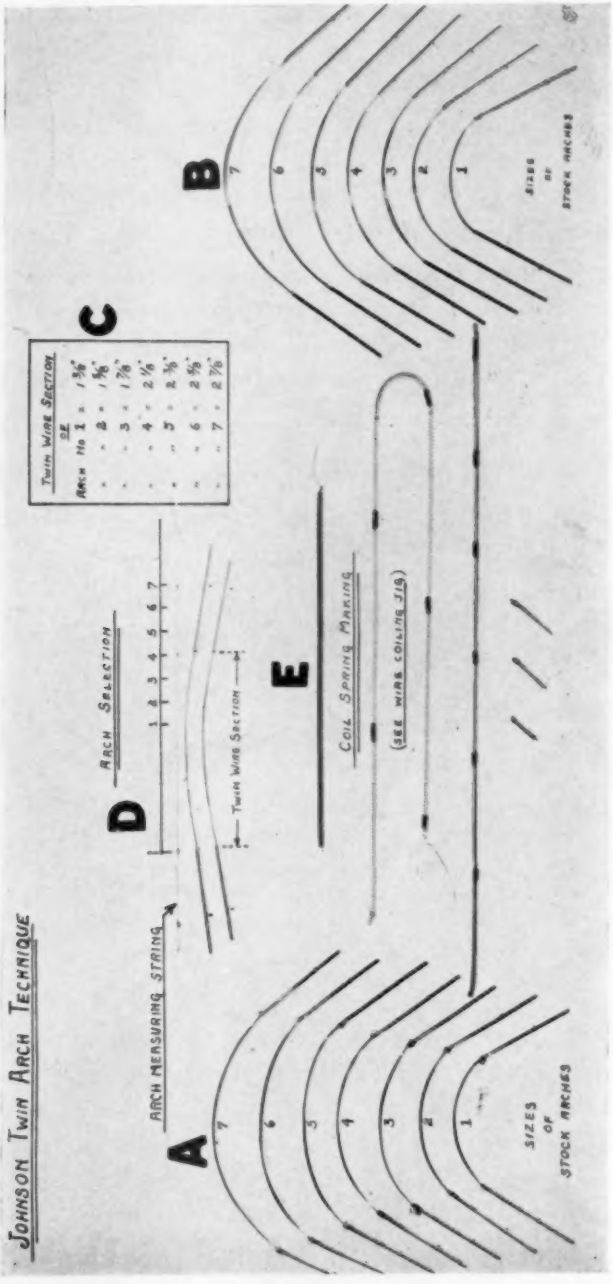


Fig. 4.

wire in the tubes is cut off. As a general rule not over three-eighths of an inch will be cut off of a tube made 1⅛ inches long. If a looped wire is used, the loop may be easily cut off by mistake, resulting in the twin wires slipping out of the tubing.

One of the geatest advantages of this vise is that the position of the intermaxillary hooks can be controlled. The twin wires should be placed in the vise with the crimps pointing forward in a horizontal position as shown in Fig. 1, *F*. The left hook should be placed a little back from an upright position and the right hook a little forward from the upright position. (See Fig. 1, *F*, and also as placed in vise in Fig. 3.) I do not understand just why this position makes the hook pull into the right position in the finished arch, but for some unknown reason it seems to work out that way.

If one hook should be slightly out of the correct position the hook can be turned by twisting the arch in the hands. This cannot be done when a recurved wire is used in place of pulling two single wires as shown. The vise shown in Fig. 3 allows this control of the positioning of the buccal tubes.

Fig. 5 shows the method of measuring for the selection of twin arches to be used. Pieces of silk ligature about eight inches long are cut and a loop is tied in one end. Such pieces are kept in the dental cabinet ready for immediate use. By looping one end over one of the buccal tubes on the molar band, the ligature is held in place and drawn around to the buccal tube on the opposite side of the

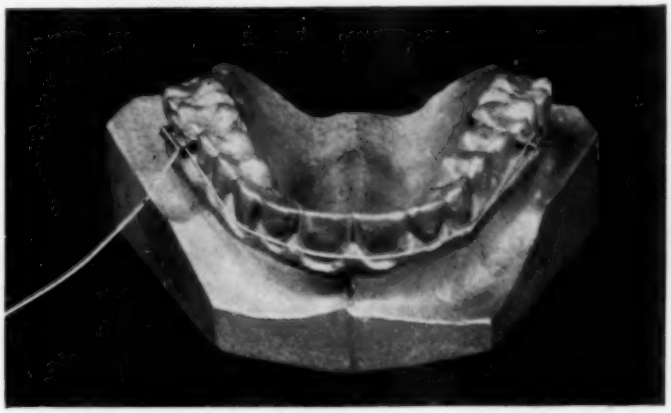

Fig. 5.

mouth. A skin or eyebrow pencil is used to mark on the string the mesial ends of the molar buccal tubes. Marks are then placed on the string at the position which is desired for the beginning of the anterior twin wire section. These marks will usually be in about the cuspid position.

The string is now taken from the mouth and given to the chair assistant who selects the desired size of arch. The string will appear as shown in Fig. 4, *D*. The two center black marks give the size of the anterior twin section and the two outer black marks indicate the position of the anterior end of the molar band buccal tubes in the patient's mouth. The Johnson side tubes are then cut a quarter of an inch beyond the outer marks. This gives a quarter of an inch of tubing to slide into the molar buccal tubes. This method allows an easy selection of one of the stock twin arches and also gives a definite measurement for the cutting of the end tubes. It eliminates all necessity for trials of the arch in the mouth, for additional lengthening of arches, and fittings for the cutting of the end tubes. We have found this method to be very efficient and a great time saver.

Fig. 6 shows the method of making coil springs. The same device as shown in Fig. 2 is used, except that the wire cutting spool has been removed. A piece of tubing (about 0.038) is run through the post parallel to the long axis of the large spool. Soldered at right angles to that tubing is another piece of tubing through which the hard-drawn wire is run.

An 0.032 wire is soldered to a hand-piece mandrel as shown. On one side of the mandrel is soldered a short piece of 0.020 tubing (approximately).

The 0.032 wire from the mandrel is now run through the tubing in the post. The hard-drawn wire for coil spring making is run through the forward tubing and the end of the hard-drawn wire run through the small 0.020 tube on the side of the mandrel. This will hold the wire when starting to coil the spring and will allow an easy release of the spring after it has been coiled, as the end of the hard-drawn wire then merely slides out of the 0.020 tube.

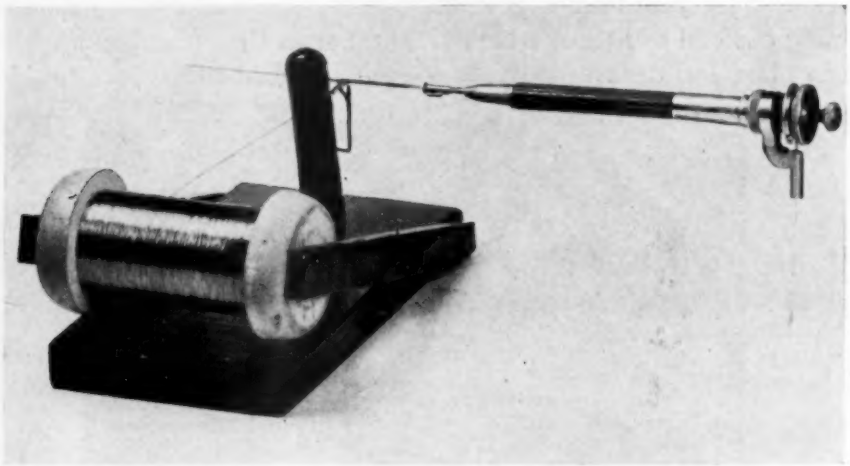

Fig. 6.

The dental engine turns the 0.032 wire winding the hard-drawn wire accurately and quickly into a coil. The forward tube controls the position of the hard-drawn wire regardless of where it may be coming from the large spool. The 0.032 wire gradually comes out of the post tubing as the coil is being spun. The tube in the post steadies the 0.032 wire so it does not bend, and also the operator does not have to hold the coil in the fingers, as this process becomes very warm if held in the fingers while spinning a coil.

When the coil comes to the end of the 0.032 wire it will automatically break off, and if the forward tube is small enough, the kink made when the wire breaks will catch on the end of the tube and prevent the wire on the large spool from uncoiling. The side pieces holding the spool should have some tension to keep the spool from unwinding too rapidly. Round head screws on the inside of the side pieces rest in the holes on the end of the spool. Making the side pieces of thin hard wood will allow them to be sprung sidewise sufficiently for changing spools.

While coiling a spring it is best to hold the spool with the hand to add tension. This will give a very tightly wound coil.

The coil is then stretched as shown in Fig. 4, *E* (U-shaped section). It should be pulled out leaving intermittent sections of tightly wound coil as shown. The stretched coil is then replaced on the 0.032 wire and tightly compressed. This will give a coil as shown. This piece is then cut into small coils of the desired length to use. One end of each coil should be left tightly wound for about a sixteenth of an inch. This tight section will fit closer to the arch tubing and will allow the springs to be stopped at the desired position on the tubing by pinching the tubing as has been demonstrated by Dr. Johnson.

The vise shown in Fig. 3 is made on order by the H. Wibling Tool & Mfg. Co., of 116 Walker Street, New York City.

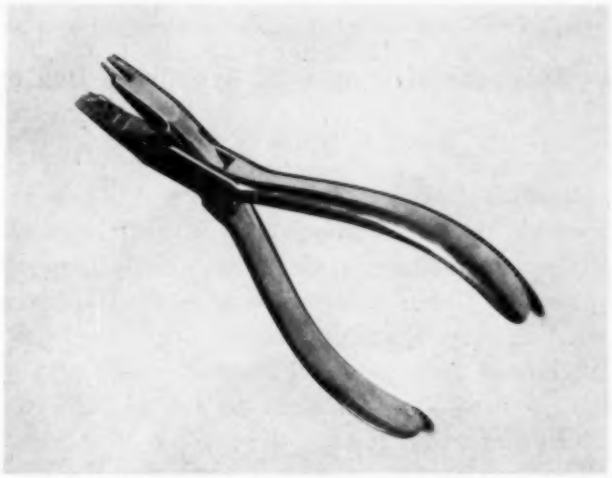

Fig. 7.

Fig. 7 shows the pliers used for making the crimps in the twin wires. This plier was made especially for Dr. Ralph Waldron of 549 High Street, Newark, N. J. Any reader desiring information about this plier should write to Dr. Waldron.

The writer wishes to express his appreciation to Dr. Johnson for the development of a very useful appliance for certain cases and only submits this clinic as an adjunct to the finely developed technique of Dr. Johnson.

41 East Fifty-Seventh Street

# Department of Orthodontic Abstracts and Reviews

Edited by

DR. J. A. SALZMANN, NEW YORK CITY

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City.

**Roentgenographic Diagnosis of Congenital Syphilis in Unerupted Permanent Teeth:** By Bernard G. Sarnat, M.D., Isaac Schour, D.D.S., Ph.D., and Robert Heupel, D.D.S., Chicago, *J. A. M. A.* **116**: 2745-2747, June, 1941.

Hutchinson described the screwdriver-shaped and notched permanent maxillary central incisors as the most common and characteristic clinical dental abnormality in syphilis. A roentgenographic diagnosis of unerupted Hutchinson incisors has also previously been reported by others. The presence of Hutchinson incisors does not necessarily mean that the permanent first molars need be affected. The Hutchinson incisor owes its characteristic shape to a disturbance which occurs early in dental development in congenital syphilis. This is at the period of morphodifferentiation when the pattern of the dentino-enamel junction (and the future crown of the tooth) is being established. The apposition of enamel which occurs later follows the pattern of the dentino-enamel junction and proceeds normally. Clinically the enamel surface is smooth.

The affected teeth of the patient described show alterations in two different phases of dental development, depending on the developmental stage which obtained at the time of the disturbance. The first is one of disturbed differentiation, in which the basic morphologic pattern of the tooth is affected, giving a convergence of the proximal surfaces of the permanent incisors and molars. Only these teeth are affected in the manner described because they are in the stage of morphodifferentiation at the neonatal period and in earliest infancy, when congenital syphilis is probably most active. Thus the permanent maxillary lateral incisors are normal because their development occurs at a later period (ten to twelve months).

The second stage is one of hypoplasia or disturbed apposition in the enamel of the deciduous teeth. These teeth are still active in the formation of enamel and reflect the disturbance of this structure. They do not show disturbed morphodifferentiation because this stage is completed before the neonatal period.

An early roentgenographic diagnosis of congenital syphilis on the unerupted permanent central incisors and first molars of a negro girl, aged 4 years, was made and confirmed clinically two years later when the teeth appeared in the oral cavity. Clinical examination of the teeth in 1940 showed a constriction of the occlusal surface. The cusps were worn, flat, and close together. Roentgenograms taken at this time were similar to those taken in 1938 but showed the

growth, calcification, eruption, and attrition that had since occurred. A comparison of the normal permanent second molar with the affected permanent first molar showed distinct differences in the contour and size of the crown. The roentgenographic diagnosis of congenital syphilis on the basis of the abnormalities of the permanent incisors and molars made in 1938 was thus confirmed by clinical examination in 1940.

The teeth showed disturbances in the developmental phases which occur during the neonatal period and earliest infancy. The effects were different in the growing deciduous and permanent teeth, depending on the developmental stage active at the time. The deciduous teeth, active in the formation of enamel, showed hypoplasia; the permanent teeth, active in morphodifferentiation, showed a disturbed dentino-enamel junction with a resulting characteristic distortion of the crown.

The conditions of the teeth are permanent and not subject to change as they are in the bones. Roentgenograms of the unerupted permanent teeth as well as of the long bones should be used as an aid in the early diagnosis of congenital syphilis.

**The Stage for Intervention in Certain Malocclusion:** By Lilah Clinch, *British Dental Journal* **70**: 121-130, 1941.

Since we are unable to predict the course of development of any individual, we should approach malocclusions in the early stages of the dentition with a bias against mechanical interference. We find that even the results of gross interference with normal development, as in thumb-sucking, may disappear, and that even gross early disproportions of growth may make a near approach to normal proportions; and we know that unless the growth of our patients' faces has that urge toward the normal, our appliances can accomplish but little at an early stage that will be of permanent benefit. Our attitude should be that unless there is a clear-cut reason for mechanical interference in the particular case under consideration, growth should be allowed to do as much as it can, while we stand aside and watch progress.

*Reasons for early interference.*—First—Temporary disproportions of growth of local factors such as retained deciduous teeth, produce tooth relationships which themselves become an obstacle to the normal progress of the dentition. Locking of one or two incisors in wrong relationship with their opponents is also a common example. These should be corrected as soon as they are clearly established, which will generally be in the early stages of the mixed dentition. Second—Wrong arch relationship in the anteroposterior direction in certain cases is more readily dealt with at an early stage than later. In those cases in which the development of the maxillary arch is approximately normal, there may be a case for early intervention because (1) the wrong incisor relationship, once established, is very unlikely to correct itself, even in the event of growth restoring normal jaw proportions; (2) the wrong tooth relationship tends to accentuate the jaw deformity; and (3) the restoration of correct tooth relationship at an early stage tends to bring about a better adaptation of the occlusion to a persisting abnormal jaw proportion than could readily be made at a later stage by mechanical means. Third—Where closed bite can be dealt with when

the first permanent molars are still in active eruption. Once the permanent first molars are in contact, there is little, if any, prospect of successful opening of the bite until the stage of eruption of the second permanent molars, so that treatment is best deferred until that stage. This applies to primary or developmental closed bite, but not to secondary. (That which is not due to a defect of growth, but to a change of tooth relationship, such as frequently occurs following premature loss of mandibular first deciduous molars.) Fourth—In certain cases in which the deciduous molars and incisors have been extracted, the mandible swings upward as the deciduous canines are not able to withstand the bite, producing an anterior position of the mandibular teeth. This can be prevented by the use of plates which provide a resting surface for the jaws in normal relationship. Fifth—When there is rotation and overlapping of incisors due to lack of space, these are not only much more difficult to retain in corrected position when treated some years after eruption than when treated while the tissues around the roots are still in process of modification, but also are liable to become carious owing to the stagnant food debris areas created by the irregularity. Even though in a proportion of these cases reduction of the number of teeth may eventually be advisable, it is valuable to correct the irregularity within six months or so of its establishment. It is not wise to assume a permanent overlap from the absence of adequate space at the time of the eruption of the first six incisors; many irregularities arising from this cause correct themselves with the spurt in growth which follows incisor eruption instead of preceding it, and with the action of the tongue. Sixth—There are cases in which disproportion of growth has been so great that it seems out of the question that a reversal of the proportions can make up the discrepancy, or where pathologic conditions exist which clearly make the normal unattainable.

*Intervention at Later Stages:* The question of the stage to which nonintervention should be carried where malocclusion persists is best approached in this way: Successful treatment of malocclusion depends mainly on the existence of normal size and normal proportions in the facial framework. If that condition is fulfilled, redistribution of the teeth can be carried out within a long period. Final size and proportions of the face are not attained until the adult stage is reached, but the growth which affects the part of the mouth anterior to the third molars is very nearly complete by the time of eruption of the second molars. Any marked growth that might correct a previous disproportion is not to be expected after that point; and even the disproportion which commonly becomes pronounced at a later stage—excessive forward growth of the mandible—has almost always declared itself by that time.

No case can be regarded as certainly complete before the third molars have been erupted, but most corrected cases can be looked on as almost certainly stable if they have been stable for a year after the eruption of the second permanent molars, without artificial retention. We have the alternatives of commencing treatment early and carrying retention up to the time of eruption of the second permanent molars, or timing the start so that completion shall coincide roughly with the completion of eruption of the second permanent molars. The first course will generally involve us in a longer period during which appliances must be worn, and it involves the use of retention appliances through

a period of approximately two years during which only the eight incisors remain in the same relative positions. Retention appliances extending beyond the incisors have either to be altered drastically and repeatedly or they interfere with the normal developmental movement of the teeth. The second course will generally involve commencing treatment at or after the time of eruption of the first premolars, and since the appliances used for active treatment are themselves designed for altering the relative position of the teeth, the difficulties that arise with retention appliances at this stage affect active treatment very much less acutely.

So that whether we look at the question from the point of view of wishing to know what is the real nature of the malocclusion with which we have to deal, or from the point of view of wishing to keep the period of wearing appliances and the trouble of maintaining appliances to the minimum, we find strong indications for undertaking treatment at the stage when the permanent dentition is taking shape.

Friel has pointed out the advantages of beginning treatment as soon as possible after a "filling out" period, rather than after a "springing up" period. (Springing up period of active growth in height, from birth to one year, from five to seven years and from eleven or twelve to fifteen years; alternating with filling out periods, from two to five years, seven to eleven or twelve years, and fifteen to twenty years.) It is during the filling out periods that the orthodontically useful growth occurs in the face. Clearly the earliest period at which grounds exist for forming a definite idea of the final type of base is at the time of establishment of premolars, canines and second permanent molars.

# Editorial

## The Pacific Coast Symposium on Orthodontics

ON FEB. 21, 1941, at the Nineteenth General Meeting of the Pacific Coast Society of Orthodontists an Educational Symposium was presented in the form of a round table discussion pertaining to some of the causes of orthodontic failures. Elsewhere in this issue of the Journal appear the papers of this session, which record a liberal education on physical diagnosis as it is related to the orthodontic problem. The subjects discussed at the meeting were as follows: Bone by Dr. John A. Marshall; Congenital Syphilis by Dr. Harry Templeton; Allergy by Dr. Henry Stafford; Ductless Glands by Dr. H. Clare Shepardson; Internal Medicine by Dr. S. P. Lucia; and Orthodontics and Growing Up, a Pediatrician's Viewpoint by Dr. Clifford Sweet—all medical men of wide and varied experience in the research problems of scientific medicine. With the observation that "I want to leave with you this word," Dr. Sweet said that, "the child who has an orthodontic deformity generally has a deformity that extends through his whole body." He thereby sharply struck the keynote that ran through the entire discussion. When he observed further in his talk in speaking to the orthodontists that "you are beginning to ask why these conditions which you are called upon to treat are present and whether or not you are meeting them in the most adequate manner," he, no doubt, put his finger exactly upon the inspiration which was responsible for the Program Committee arranging for this new and interesting part of the program.

The orthodontic specialty plainly originated in the beginning on account of an urgent demand for a means of correcting unsightly malocclusion of the teeth, a deformity so prevalent in growing children. Now after years of experience by many workers in the field there appear steadily more and more curiosity and bewilderment in regard to some of the unsolved problems and speculative manifestations met in everyday orthodontic practice.

In the beginning treatment was based almost entirely on the premise of mechanical dexterity. The etiology, according to early textbooks and manuscripts, was confined largely to local causes such as thumb-sucking, early extraction of deciduous teeth, mouth-breathing, and many others. Heredity was dismissed with a gesture, constitutional causes were, and are, not well understood among workers and writers. So now it is only a natural sequence of events that orthodontists should seek the field of scientific medicine in order to help them solve some of the important questions still unsolved in their field.

The mechanical era of orthodontics made spectacular progress and its creditable corrections stand out as some of the most outstanding contributions made in the advance of dentistry, notwithstanding many orthodontists of wide experience obviously feel that the field in which satisfactory correction may be anticipated in advance of treatment by mechanical means alone is restricted

and has borderline limitations indeed. It can be said at least with quite some degree of security that the fine record of orthodontic cases that have turned out satisfactorily during the past years has not been established in that class of cases in which the etiologic factors might be classed as constitutional in character. It seems, therefore, logical to assume that the time has long since arrived when the strictly mechanical viewpoint based upon the assumption of the constancy of the position of the maxillary first molar or the fictional application of the orbital plane, and their application to orthodontic practice, will suffice for all of the problems in hand. While not to be discounted in importance, at the same time these concepts must move over and make room for a more comprehensive and better basic foundation for the subject, at least from the standpoint of etiology.

Much fine work has been done in the endocrine field that has added much to the field of orthodontic diagnosis. Much is now on the way in the field of heredity. Sign posts ahead indicate many other constitutional disorders are being slowly linked up with the problem of dental anomalies that promises developments that will add to the grand total of the knowledge of the whole problem.

In the composite orthodontic problem the mechanical viewpoint is important because it still requires a jeweler's touch to make a good orthodontist, and always will. The anatomic viewpoint is essential because without it the normal is a speculative and unknown; however, we are learning that nutrition, heredity, and constitutional disorders have much to contribute to the problem at hand.

The California Symposium makes interesting reading for many practicing orthodontists who have been baffled and bewildered by the lack of response of a certain per cent of their cases. Perhaps a part of the answer to these problems may lie in the field ahead, that of scientific medicine. It will at least serve the purpose of making the orthodontist nutrition-minded so that he will realize the border line or limitation where mechanical therapy and the theory of the constancy of the first molar or other orthodontic fictions related to diagnosis leave off and the broader perspective begins.

In between the lines of the California Symposium may be read inferentially two very interesting stories. On the one hand is written much of the story of the so-called "perpetual care" orthodontic cases (well known to orthodontists); on the other hand may be gleaned much of the inside story behind what is obviously a part of the inspiration, at least, for the new so-called Harvard plan of dental education, the plan that proposes to teach dentists more of scientific medicine than they have known in the past. Orthodontists as a group obviously are conscious of their limitations in this field.

*H. C. P.*

# Special Feature

## PRESENTATION OF THE ALBERT H. KETCHAM MEMORIAL AWARD, 1941, BY HARRY E. KELSEY

MR. PRESIDENT, Dr. Noyes, Members of the American Association of Orthodontists and of the American Board of Orthodontics:

If there are any trying duties which the president of the American Board of Orthodontics is called upon to perform, this is not one of them. It will be a matter of lasting satisfaction to me that my term of office as president of the American Board did not expire until I had the pleasure of presenting to you as the recipient of the Ketcham Memorial Award the name of my old and very good friend, fellow classmate and teacher at the Angle School of Orthodontia, Dr. Frederick B. Noyes. My satisfaction in performing this duty is not alone because of the personal pleasure it affords me, but also because of the fact that the honor in this, as in past instances, is so markedly deserved; I may add that the difficulty of preparing a brief narrative account of the life of a man who has wholeheartedly given fifty years of distinguished service to his profession is not in finding material to dwell on, but in trying to select from the voluminous record of his accomplishments those events which seem most appropriate for an occasion of this kind.

Although I had known of Dr. Noyes in the field of histology, I first met him personally in 1908 when I went to New York to take the course at the Angle School of Orthodontia. Since 1904 he had been lecturing in the school on histology and embryology and had decided that year to take the course. In this way I became his classmate and pupil and in a less active way I have remained his pupil ever since. Those of you who attended the Angle School will remember with what trepidation prospective candidates went in to take their preliminary tests; and this after months of preparation. I know in my own case that after more than ten years in general practice I was, in proposing to give it up, playing for big stakes, and as I never had a good poker face, it must have been apparent that I was just plain scared. His genial and kindly reception soon put me at ease and restored my determination to succeed in the struggle to crowd a year's work into the short two months' course that was to ensue. He asked me about my own work and seemed to be aware of one or two small contributions I had had the temerity to attempt in the field of orthodontics, and from that to other topics that might under other circumstances have been of mutual interest. It was a pleasant meeting and one that I have never forgotten. As I was going out the door I almost (but not quite) turned back to remind him that he had forgotten to examine me. Perhaps he had without my

knowledge already done it. I speak of this to show the innate kindliness of the man and his capacity for human sympathy. Were there time, many similar incidents could be recounted occurring throughout his long professional life.

Endowed to a marked degree with those unselfish qualities which inspire the possessor with a desire to share his success and good fortune with others, Fred Noyes not only did not miss, but definitely made opportunities to aid and develop younger men and inspire them with the same high ideals which have been such a strong governing influence in his own life. He had rare ability in this respect, and it has borne fruit as witness the names of Isaac Schour, J. R. Blayney, Allan G. Brodie, Robert Kesel, Burne O. Sippy (whose death was a real loss to the profession), and, of course, his son, Harold J. Noyes.

Few men have had a finer background or more impressive opportunities and fewer still have made such conscientious use of them. Such things imply an obligation which Dr. Noyes has been discharging and will, I am sure, continue to discharge for years to come to the advancement and elevation of his profession. Of course, his parents constituted the earliest and probably the most important component in that background. There are doubtless many here who, like myself, had the pleasure of knowing Dr. and Mrs. Edmund Noyes, and who will, I am sure, agree with me that here was the first great influence on Dr. Noyes' life as a man and as a member of the profession which his father served for so many years in such an honorable and honored capacity. Dr. Edmund Noyes was one of the outstanding figures in his profession during a long and useful career.

Then came his long and close association with Dr. G. V. Black. I asked Dr. Noyes to give me some account of this and am taking the liberty of quoting from his response, "In regard to my relation with Dr. Black my first contact with him was about this time in the spring of 1888 or 89. I am not sure which. Black had lived in my father's house during the winter while he was giving all his time to teaching in the Chicago College of Dental Surgery. It had been a very trying winter for him. When I came home from Beloit Academy for spring vacation, he was on the verge of a breakdown. Father called up a little resort hotel that he knew of on Fox Lake, found that the ice had just gone out of the lake, and arranged for Dr. Black and me to spend a week there.

"Even before I graduated in 1895 until his death I was closely associated with his research activities. In his study of the amalgams he wanted a check on his amalgam micrometer and got me to examine the tubes for shrinkage or expansion. After he had measured them he would make the reading of the micrometer, record it, and hand the tube to me. I would examine it with a binocular microscope and record it. By judging the width of the shrinkage crack or the extent that the filling had elevated above the steel, I found I could check his measurements within the fraction of a one-thousandth of an inch. I assisted Dr. Black in many of his demonstrations before dental societies in this country and Canada.

"Since I graduated in '95 Dr. Black got me to organize a course in dental histology, based very largely on his series of articles on the structure of the

teeth in the *American System of Dentistry* and the articles published in the *Dental Cosmos* about that time. After one year they gave me both the general and the dental histology. Of course in planning the details of this Dr. Black and I worked together continuously.

"In most of the articles which he published I made the photomicrographs to illustrate them, and from 1900 until 1908 we spent nearly every Sunday during the winter working on the illustrations for the operative dentistry and the special pathology. I would take the Illinois Central train at 57th Street and he would get on the same train at 47th St. We would go down to the college and work all day. Naturally this was considerably better than a graduate course."

This long and close association with Dr. Black could not but have a fine influence on the character of a young man as well as on his professional growth and development.

Then came the influence of the Edward H. Angle School of Orthodontia. In building up his unusual list of instructors for the school, Dr. Angle was attracted by the work of Noyes in histology and embryology and after agreeing to teach these branches Dr. Noyes threw himself into his work with the characteristic enthusiasm which had marked all his previous professional endeavors.

Of the many honors which have come to Dr. Noyes, I am sure one that gave him, and all of his friends, as much pleasure as any was the dinner given in his honor in recognition of his service to dental education and the dental profession by the Faculty of the University of Illinois College of Dentistry, which was attended by such outstanding men as Arthur Cutts Willard, President of the University of Illinois, Leroy M. S. Miner, Past-President of the American Dental Association and Dean of Harvard University Dental School, Isaac Schour, President-Elect of the International Association of Dental Research, Arthur H. Merritt, then President of the American Dental Association, and The Reverend R. Everett Carr, Rector of St. Peter's Episcopal Church of Chicago, who were assigned to speak on various phases of Dr. Noyes' life.

President Willard spoke of the value of Dr. Noyes to the University of Illinois saying, in part, "I think Dr. Noyes has as high professional ideals as any one I know. Unless professional ideals are kept upon a very high basis both ethically and scientifically, they do not merit the respect of a true profession. Dr. Noyes personifies the highest of professional ideals and their application. I know that with great energy, Dr. Noyes, you will continue to stand for the highest ideals of the profession of dentistry and to help place dentistry as high as any recognized profession."

Dr. Miner told of the respect that the deans of other dental schools held for Dr. Noyes' judgment and remarked that his council would be sorely missed.

Dr. Merritt referred to the influence of Dr. Noyes upon young men inspiring them with the same qualities of heart, mind, and devotion to duty which are so characteristic of him.

Dr. Schour spoke of Dr. Noyes, the scientist, referring to the fact that, "His book on *Dental Embryology and Histology* has the rare distinction not

only of being the first text ever published on this subject but also of maintaining its position as the standard text for a period of nearly thirty years, in spite of the fact that a number of new texts have appeared since the first edition of his text in 1912."

Dr. Brodie spoke of the contributions of Dr. Noyes to orthodontics, and The Reverend R. Everett Carr paid tribute to the value of Dr. Noyes to his community and to his church.

Time does not permit further comments on the achievements of Dr. Noyes. They have already been more ably recounted in the program of the dinner given in his honor and in the report of it published in the Alumni Bulletin of the University of Illinois College of Dentistry. However, conforming to custom, some brief historical notes and mention of a few of his most notable contributions to the profession are appended. "Frederick Bogue Noyes was born in Chicago on Aug. 22, 1872. His father, Dr. Edmund Noyes, had been practicing dentistry in Chicago since 1868. On his father's side he was descended from the Noyes who came to Massachusetts from England in 1632. In England the family was of Norman-French origin, a Noyes receiving a grant of English land in the Doomsday Book. His mother was English, coming to this country when she was a young girl."

1. Dr. Noyes was: One of the first to use microphotographs and develop microphotography in dental fields.

2. He was intimately associated with Dr. G. V. Black and was responsible for many of the illustrations in his book, *Operative Dentistry and Special Pathology.*

3. One of the first in this country to organize seriously the teaching of dental histology. The first edition of his text on that subject was one of the earliest devoted exclusively to dental histology.

4. Included in his histologic studies, the investigation and report on the lymphatics of the teeth and oral structures was an important and original contribution.

5. While by no means the first, he was one of the earliest specialists in orthodontics in the Middle West, a contributor to the literature of the specialty as well as its developmental progress.

6. He assisted in originating the following dental societies: American College of Dentists, charter member; Association for the Advancement of University Education and Dentistry; International Association for Dental Research; Odontographic Society of Chicago, charter member.

7. An ardent advocate, in fact as well as in fancy, of a firm foundation in the basic sciences as essential in the building of dentistry to a science and profession rather than a mechanical trade; he has been of important service to American Dental Education in general and the Dental Department of the University he has served in particular. There the educational program, as well as the very building the Department occupies, owe their being to his vision, determination, and perseverance.

Dr. Noyes has the following degrees: A.B., Johns Hopkins University, 1893; D.D.S., Northwestern University Dental School, 1895; L.L.D., Temple University, 1938; Sc.D., Northwestern University, 1922; Angle School of Orthodontia, 1908.

He was Professor of Histology, Northwestern University Dental School, 1896 to 1914; Professor of Histology and Orthodontics, University of Illinois College of Dentistry, 1914 to 1917, Professor of Orthodontics, University of Illinois, College of Dentistry, since 1917, and Dean, 1926 to 1940.

He received the Callahan Medal award by the Ohio State Dental Society, 1922, the Jarvey Medal awarded by the New York State Dental Society, 1929, and was a charter member of the American College of Dentists.

He is a member of Kappa Alpha (Southern); Denta Sigma Denta; Omicron Kappa Upsilon; Sigma Xi, Chicago Dental Society; Illinois State Dental Society, President, 1922; American Dental Association; Institute of Medicine of Chicago; Fellow of American Medical Association; Chicago Association of Orthodontists, President, 1927; Honorary Member, American Association of Orthodontists; Odontological Society of Chicago; Odontographic Society of Chicago, President, 1903-04; International Association of Dental Research, President of Chicago Section 1920-28.

He is the author of a textbook of dental histology and embryology which is in its fifth edition. His papers and journal publications too numerous to list here have been chiefly on histology, orthodontics, and education.

And now, Dr. Noyes, as President of the American Board of Orthodontics, I have the high privilege of handing to you this parchment on which is inscribed, "The honor signalized by the Albert H. Ketcham Memorial is hereby conferred upon Frederick Bogue Noyes. This award is annually made in recognition of valuable contributions to the science and art of orthodontics." It is the tangible evidence of this award and I am sure if our former friend and confrere, Albert Ketcham (in whose honor this memorial was established), could be with us today, he too would be as happy as are we over your selection for this honor.

833 Park Avenue

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## RESPONSE TO THE PRESENTATION OF THE ALBERT H. KETCHAM MEMORIAL AWARD BY DR. FREDERICK B. NOYES

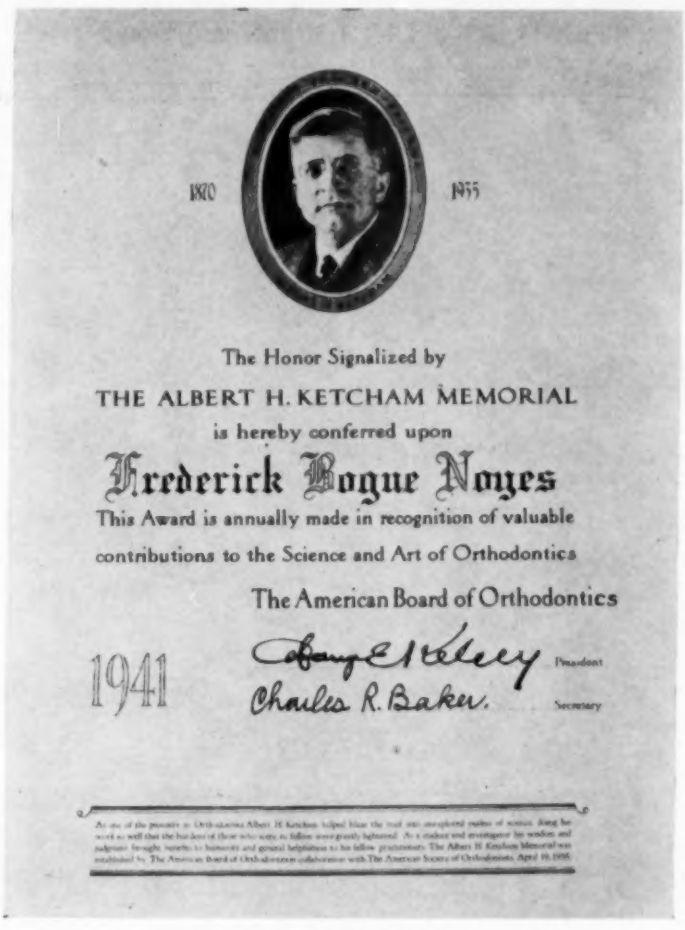
Mr. President, Dr. Kelsey, and Members of the American Association of Orthodontics:

In accepting this award, I am most deeply grateful, although conscious that I little deserve it, and keenly appreciative of the recognition and the distinction that you have bestowed upon me.

In accepting it, it is an added pleasure that it is established in the memory of a man whom I counted as one of my very best friends for many, many years. Dr. Albert H. Ketcham was a very unusual man, a man of sterling

integrity, of untiring energy, of indefatigable enthusiasm. I have always marveled at the man when I recall that he left Boston and went to Denver on a stretcher to die, and would not. I have said that if somebody came from behind Albert Ketcham and hit him on the head with an axe, he would jump up and say, "By gosh, you missed me."

His devotion to orthodontics has been one of the great factors which has led to the wonderful development of the profession in this country, and his devotion and love for the children he served has been, and always will be to me, an inspiration. He never forgot his patient. He never forgot for a moment that he was not only rendering a professional service, but was having an influence on the character of every one of those individuals. His service to orthodontics was very, very great and will continue to last.




The Ketcham Award

And now, I simply want to call on you, in the name of Dr. Ketcham, to continue the development of orthodontics as it has continued since the turn of the century. I call you to work because it is not going to advance except by your effort. And this time I want to remind you that there is an obligation not only to your profession, but an obligation to your country, and the biggest job in this country is to arouse yourselves, your neighbors, and the entire

nation to the realization of what we face. We have got to take over the work of the men who are going to be called to the service of the armed forces of the United States, and that means pulling in your belt and going to work. For my part, I would rather die working and with my shoes on than to slip from the ease of an armchair to the oblivion and uselessness of the grave.

We have got to work, and we want to remember that a year ago, in our democratic fashion, we called a man to lead this country, and we laid upon him an enormous, a horrible responsibility, for in his position as President and executive of this nation he is also the Commander-in-Chief of its armed forces. And if he is to succeed and we are to persist as a nation, we must enthusiastically, wholeheartedly, devotedly stand behind the man we have chosen for a leader for four years more.

I abominate war and hate the brutality and the abomination of it, but it is better to fight than live as a slave. Personally, I would rather die fighting than live in a world dominated by the arrogance and the brutality of a victorious Germany. So I call you to work for your profession and for your country.

# News and Notes

## Great Lakes Society of Orthodontists

The Great Lakes Society of Orthodontists will meet in Ann Arbor, Mich., Nov. 3 and 4, 1941.

## Midcontinent Dental Congress

The Midcontinent Dental Congress will meet in St. Louis, Mo., Nov. 17, 18, and 19, 1941.

## A. D. A. Meeting

The American Dental Association will meet in Houston, Texas, from Oct. 27 to 31, 1941.

## Xi Psi Phi

Xi Psi Phi Fraternity will hold its annual reunion on Monday, Oct. 27, 1941, in the South American Room of the Rice Hotel, Houston, Texas.

DR. L. I. KERCHEVAL, Chairman
Local Arrangements Committee
1625 Main Street, Houston, Texas

## Forsyth Alumni Association

The Forsyth Alumni Association will hold its annual dinner on Tuesday, Oct. 28, 1941, at 7 P.M., in the Lacquer Room of the Rice Hotel.

DR. MARCUS D. MURPHY, Chairman
Local Arrangements Committee
2017 West Gray, Houston, Texas

Charles F. Mitchell, D.D.S., announces the opening of his office at 77 Maple Street, Springfield, Mass., for the exclusive practice of Orthodontics.

## OFFICERS OF ORTHODONTIC SOCIETIES*

### American Association of Orthodontists

*President,* Claude R. Wood _ _ _ _ _ _ _ _ 608 Medical Arts Bldg., Knoxville, Tenn.
*Secretary-Treasurer,* Max E. Ernst _ _ _ 1250 Lowry Medical Arts Bldg., St. Paul, Minn.
*Public Relations Bureau Director,* Dwight Anderson
_ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ 292 Madison Ave., New York, N. Y.

### Central Association of Orthodontists

*President,* Harold J. Noyes _ _ _ _ _ _ _ _ _ 55 E. Washington St., Chicago, Ill.
*Secretary-Treasurer,* L. B. Higley _ _ _ _ _ _ _ 705 Summit Ave., Iowa City, Iowa

### Great Lakes Society of Orthodontists

*President,* Frank S. Cartwright _ _ _ _ _ _ _ Henry Ford Hospital, Detroit, Mich.
*Secretary-Treasurer,* Richard E. Barnes _ _ _ _ _ _ 838 Keith Bldg., Cleveland, Ohio

### New York Society of Orthodontists

*President,* Sidney E. Riesner _ _ _ _ _ _ _ _ _ 136 E. 36th Street, New York, N. Y.
*Secretary-Treasurer,* William C. Keller _ _ _ _ 40 E. Forty-Ninth St., New York, N. Y.

### Pacific Coast Society of Orthodontists

*President,* Ben L. Reese _ _ _ _ _ _ _ _ _ _ _ Roosevelt Bldg., Los Angeles, Calif.
*Secretary-Treasurer,* Earl F. Lussier _ _ _ _ _ 450 Sutter St., San Francisco, Calif.

### Rocky Mountain Society of Orthodontists

*President,* A. B. Brusse _ _ _ _ _ _ _ _ _ _ _ 1558 Humboldt St., Denver, Colo.
*Secretary-Treasurer,* Robert L. Gray _ _ _ _ _ _ _ Republic Bldg., Denver, Colo.

### Southern Society of Orthodontists

*President,* Fred G. Hale _ _ _ _ _ _ _ _ _ _ _ Professional Bldg., Raleigh, N. C.
*Secretary-Treasurer,* T. C. Sparks _ _ _ _ _ _ 1508 Washington St., Columbia, S. C.

### Southwestern Society of Orthodontists

*President,* E. Forris Woodring _ _ _ _ _ _ _ _ _ Medical Arts Bldg., Tulsa, Okla.
*Secretary-Treasurer,* R. E. Olson _ _ _ _ _ _ Union Nat'l Bank Bldg., Wichita, Kan.

### American Board of Orthodontics

*President,* Charles R. Baker _ _ _ _ _ _ _ _ _ _ 636 Church St., Evanston, Ill.
*Vice-President,* Frederic T. Murlless, Jr. _ _ _ _ 43 Farmington Ave., Hartford, Conn.
*Secretary,* Bernard G. DeVries _ _ _ _ _ _ _ Medical Arts Bldg., Minneapolis, Minn.
*Treasurer,* Oliver W. White _ _ _ _ _ _ _ 213 David Whitney Bldg., Detroit, Mich.
William E. Flesher _ _ _ _ 806 Medical Arts Bldg., Oklahoma City, Okla.
James D. McCoy _ _ _ _ _ _ _ 3839 Wilshire Blvd., Los Angeles, Calif.
Joseph D. Eby _ _ _ _ _ _ _ _ _ 121 E. 60th St., New York, N. Y.

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### Harvard Society of Orthodontists

*President,* Harold J. Nice _ _ _ _ _ _ _ _ 475 Commonwealth Ave., Boston, Mass.
*Secretary-Treasurer,* Edward I. Silver _ _ _ _ _ _ _ 80 Boylston St., Boston, Mass.

### Washington-Baltimore Society of Orthodontists

*President,* Paul W. Hoffman _ _ _ _ _ _ _ 1835 Eye St., N. W., Washington, D. C.
*Secretary-Treasurer,* Stephen C. Hopkins _ _ _ _ _ 1726 Eye St., Washington, D. C.

### Foreign Societies†

### British Society for the Study of Orthodontics

***President,* S. A. Riddett**
***Secretary,* R. Cutler**
*Treasurer,* Harold Chapman

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis, Mo., U. S. A.


1907 class of the Angle School of Orthodontia, St. Louis, Mo. *1*, Wellslake D. Morse. *2*, E. H. Wuerpel. *3*, S. Y. Teraki. *4*, Walter E. Newcomb. *5*, E. H. Angle. *6*, Joseph Oldtmann. *7*, W. E. Wilson. *8*, W. C. Smith. *9*, Frederick B. Noyes. *10*, J. A. Cameron Hoggan. *11*, A. A. Solly. *12*, Roscoe A. Day. *13*, Frank E. Sheldon. *14*, George W. Grieve. *15*, John Mills. *16*, Martin Dewey. *17*, J. B. Stewart. *18*, ———. *19*, Glenn F. Bowman. *20*, A. H. Thompson. *21*, Paul W. Prewitt. *22*, C. A. Corrigan. *23*, Lawrence A. Krejci. *24*, H. D. Morehouse. *25*, Mrs. E. H. Angle. (Courtesy of Dr. B. W. Weinberger New York, N. Y.)